Semi-Annual Meeting, St. Louis, Missouri, November 29-30, 1953

VOLUME XXIV NUMBER 1

DISEASES

of the

CHEST

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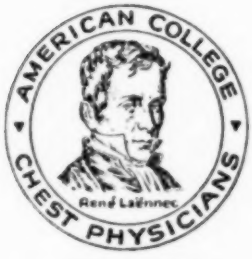

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1. Kaufman and Farmer (1951), Ann. Allergy, 9:89, January-February
2. Swartz (1950), Ann. Allergy, 8:488, July-August
3. Krasna et al. (1949), J. Allergy, 20:111, March

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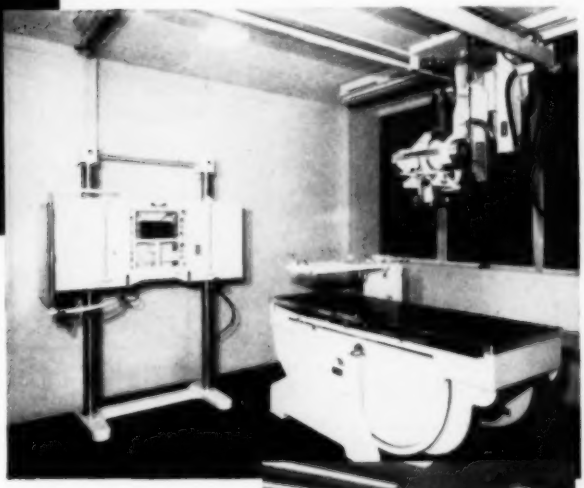

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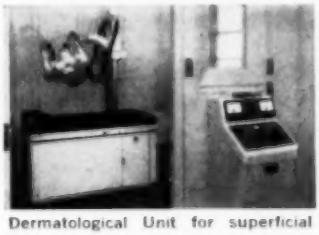

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MURRAY KORNFELD, *Managing Editor*

VOLUME XXIV JULY 1953 NUMBER 1

CONTENTS:

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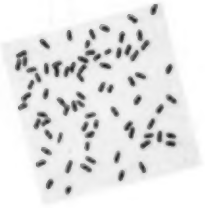

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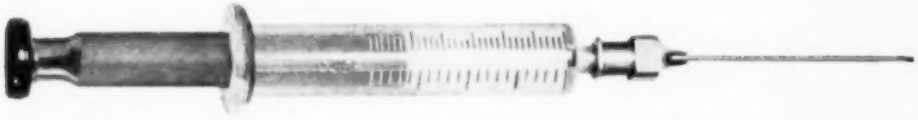

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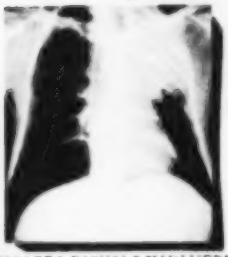

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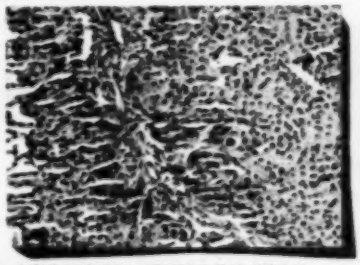

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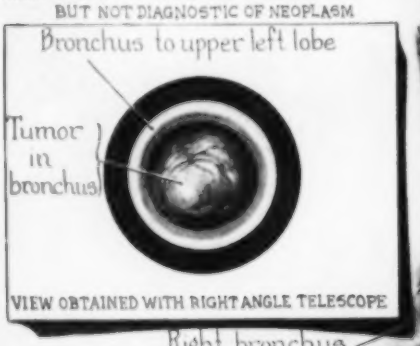

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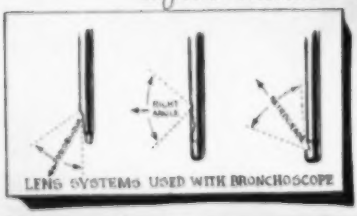

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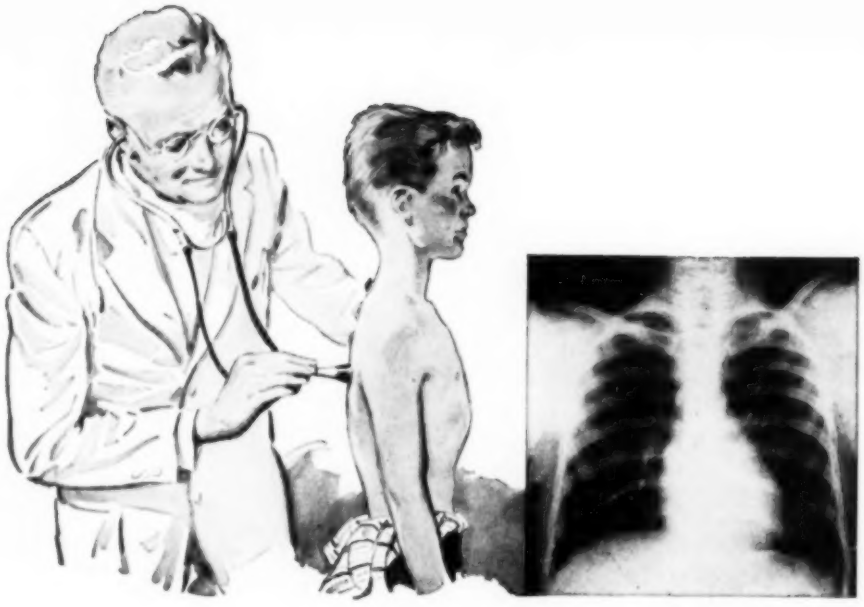

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DISEASES *of the* CHEST

VOLUME XXIV JULY 1953 NUMBER 1

Mental Hygiene in the Prevention of Irregular Discharge of Tuberculosis Patients*

FRANCIS J. WEBER, M.D., Dr. P.H., F.C.C.P.†
Denver, Colorado

I. *The Problem of Irregular Discharge*

Irregular discharge** is considered one of the most serious problems in the management of tuberculosis patients. The reports of various authors such as Tollen,[1] Whitney and Dempsey,[2] Galinsky and Brownstone,[3] Drolet and Porter,[4] as well as many others indicate the proportion of such irregular discharges in tuberculosis hospitals throughout the United States to be seldom less than 25 per cent of all patients discharged alive, and in some instances over 80 per cent. The national average would seem to fall somewhere close to 40 per cent.

The effect of this problem can be considered in terms of its consequences regarding: (1) the health of the patient, (2) the danger to the public health, and (3) the loss of public funds invested in the care of such patients. With respect to the first, Brewster and Fletcher[5] conclude from their studies: "Discharge against advice was closely related to subsequent mortality, increasing it from two to more than three times the rates for those whose discharge had been approved." The consequences to the public health are equally important. The majority of such patients still have active disease and hence constitute sources of contagion to their families and associates. Thus a good part of public health casefinding efforts is nullified through failure to keep such sources of infection out of circulation. The third factor, that is to say, the financial cost to the community, needs little explanation beyond saying that little is gained on the public's financial investment in plant, equipment and services where adequate treatment for such a large proportion of patients is rendered impossible.

Why do so many patients leave tuberculosis hospitals in an irregular manner? Much data respecting the motives that seem important have been

*Presented in part at the National Tuberculosis Association Annual Meeting, Boston, Massachusetts, May 27, 1952.

**Irregular discharge, as defined by Tollen,[1] means "any termination of hospitalization of a living patient which is not medically sanctioned by professional authority." This includes patients who are discharged against medical advice, patients who go AWOL or fail to return from temporary leave, and patients who are discharged for disciplinary reasons.

†Regional Medical Director, Rocky Mountain States, U. S. Public Health Service.

collected in an attempt to answer that question. These studies mention most frequently as motivating conditions: (1) factors in the personality of the patient; (2) factors at the institution in which he is hospitalized; and (3) factors outside the patient and the institution, mainly those in the home environment from which he came. Therefore, it is reasoned, if the problem is to be met successfully, such factors must be recognized and dealt with early in treatment in order to prevent treatment failures occasioned by premature discharge. In deciding how to go about that, Tollen[1] and his associates estimate that 70 per cent of such discharges could be prevented by individualized casework with the patient. In the point of view expressed by that author, treatment of the tuberculosis patient must include methods calculated to bring about a satisfactory personal adjustment of the patient in the hospital setting.

The study described in this paper is concerned with the latter aspect of tuberculosis treatment. In this work an attempt was made to determine, with the aid of certain statistical controls, the degree of effectiveness of some simple mental hygiene technics such as discussion, explanation, education, re-education, suggestion and reassurance employed along with personality study, in the furtherance of adjustment of tuberculosis patients in an actual hospital setting.

II. *The Data and Their Treatment*

The data of this study are derived from interviews conducted by the author with patients admitted to the State of Maryland Tuberculosis Hospital at Henryton, Maryland. The patients studied number 75 first admissions admitted consecutively during the period July 1, 1950 to March 26, 1951. All were male Negroes, 16 years or older, representing various stages of pulmonary tuberculosis (Table I).

The interviews were conducted in such a manner as to make possible the collection of uniform data respecting the patient and, at the same time, through the technics enumerated, treat any problems that arose. Through

TABLE I

The composition of the study group with respect to age and state of disease on admission

Age	Minimal	Moderately Advanced	Far Advanced	Total	Per cent of Total
16-19	3	1	2	6	8.0
20-24	0	7	2	9	12.0
25-34	0	2	6	8	10.7
35-44	3	8	16	27	36.0
45-54	1	5	9	15	20.0
55-64	1	3	4	8	10.7
65 and over	0	1	1	2	2.6
TOTAL	8 (10.7%)	27 (36.7%)	40 (52.6%)	75	100.0

discussion with the patient combined with direct observation, it was possible to construct a simple biography concerning his course of personal, educational, vocational, social, and physical development, in addition to some knowledge of particular stresses encountered in pre-morbid life, and an estimate of certain attitudes that appeared during the interview work and in the general hospital setting.

The method followed in this study in the conduct of interviewing was that commonly called "counseling," or "psychotherapy," an approach well described by other workers.[6,7] In this approach, the physician or counsellor attempts to create an informal atmosphere calculated to encourage the patient to talk freely. It is, therefore, patient-centered from the first and contains a large element of emotional catharsis for the patient. In that way the interviewer is most likely to learn what problem or problems concern the patient at a given time.

In the work done here, the first problem usually encountered was one having to do with the patient's "case" of tuberculosis. This provided the springboard of discussion from which the physician could begin to learn what the disease actually meant to the patient. As this problem was tackled together, through the technics set forth, it was found possible to help the patient gain a realistic conception of what tuberculosis is and what he could do to assist in the program of recovery. Other problems that arose from time to time were managed in the same way.

The first interview with each patient was held as soon as possible after admission. Each interview was one half to one hour in length, but in no case did interviews total more than two hours in any one month. The actual time given the patients in this study group averaged 1.3 hours per patient per month.

This work was conducted independently of the rest of the hospital staff in the sense that no attempt was made to change staff practices, administrative procedures, or to increase other personnel services that might influence one way or another the problem of irregular discharges.

III.I *Generalized Results and Their Interpretation*

A. Outcome of Hospitalization

The present section is concerned with (1) an evaluation of the results of the procedures described above in the attempt to assist patients to make a satisfactory adjustment to hospitalization, and (2) the presentation of a brief analysis of other data secured during the course of interviews with patients in the study group.

The tabulation in Table II shows the experience of the 75 patients admitted to the study group during the nine-month period of study.

Of the two discharged irregularly, one of them left against his will following an administrative decision of a physician in temporary charge, a discharge that could have been prevented through a more judicious application of the rules. It therefore involved administrative judgment but was, nevertheless, counted as an irregular discharge. The other was a 63 year

old man who failed to show evidence of active tuberculosis while in the hospital, was convinced he did not have the disease, and did not return from a holiday pass.

TABLE II

The experience of male patients with reinfection tuberculosis admitted to the Henryton Hospital and treated during the period July 1, 1950 to March 26, 1951

Outcome	Number
Discharged alive with consent	6
Discharged irregularly	2
Died in hospital	15
Remaining in hospital at close of study	52
TOTAL	75

B. Interpretation of the Data on Outcome

A problem of this kind is difficult to evaluate in terms of concurrent controls. First of all, there is the time factor. In a long term disease like tuberculosis, a considerable period of time is required before an evaluation is possible, plus the fact that a large number of patients is required. To do this would tax the resources of one person engaged in such a study and would be impossible to do within the prescribed time limit. Another difficulty, considered by the author to be more important than the first, rests in the necessity of conducting the work in a group situation. This means that interview therapy given to half a patient population would be extremely difficult to evaluate in view of the possible effect that successful therapy might exert on other patients not under treatment but in the same setting. Indications of such effect were to be seen in the case of patients on the ward admitted during earlier periods as well as readmissions who were not taken for study. Unfortunately, there is no placebo, comparable to saline injections used in drug control studies, that could be applied to patients not receiving the interview therapy. For these reasons, concurrent controls were not thought to be advisable.

The way taken out of the difficulty here was to give treatment to every patient admitted after a certain date. It is then possible to compare the effects of this as judged by the outcome in the study group, with that of a comparable group not receiving such treatment. In that respect there was a ready group for comparison, namely, patients similar in all essential respects, admitted to the hospital during a previous period. Therefore, the group of male first admissions admitted consecutively during the period January 1, 1948 to December 31, 1949 was selected for comparison. This group comprised a total of 315 patients. The various types of outcome were noted (Table III) and the time of occurrence of such events noted and entered in the tabulation. This tabulation gives the experience of the group in successive intervals.

TABLE III

Experience of 1948-1949 Control Group

Time Period in Months (x-xth)	NUMBER OF PATIENTS — Present at Beginning of Period (Ox)	Number Withdrawing for Specified Reasons — Died (Dx)	Discharged with Consent (Cx)	Irregular Discharge (Sx)	Person-Months at Risk (Lx)	Rate of Irregular Discharge in Period Msx (Sx/Lx)
0-1	315	44	1	8	288.5	.029
1-2	262	27	2	9	243.0	.037
2-3	224	17	0	8	211.5	.038
3-6	199	27	5	21	517.5	.041
6-9	146	17	1	18	384.0	.047
TOTAL		132	9	64	1,644.5	

$$Lx = Ox - \frac{(Dx + Cx + Sx)}{2}$$

Table III was constructed as follows: The first month's experience of all 315 patients was combined regardless of the time admitted during the 24-month period. This is possible because time intervals begin with admission. After doing this, the experience of patients during the first month can be recorded as follows:

All (Ox) patients	315
Died in (Dx) this month	44
Discharged with consent (Cx) in this month	1
Discharged irregular in (Sx) this month	8

To recapitulate, this shows at the end of the first month (0-1) the number of patients who were under observation during the interval (Ox); the number of patients on whom observation terminated during the period by way of deaths (Dx); discharges with consent (Cx); and those who left irregularly (Sx).

Thus we come to the beginning of the second month with Ox now Ox−N, with N equal to the total of deaths and discharges occurring in the first month, and therefore Ox becomes 315−53 262. The experience during the second month is then represented as:

X-Xth	1-2	Cx	2
Ox	262	Sx	8
Dx	27		

The remainder of the table is constructed by continuing in the same manner and working through successive periods.

From this we see what happened to patients in that group during successive periods of time. From this data we are able to derive the rate of occurrence (Msx) of each event, such as irregular discharge, occurring within any stated period of time. A study of Table III indicates that during the early months following admission mortality is heaviest, irregular dis-

charges are also great, and as one might expect, discharges with consent are low.

By applying the Msx derived for each time period to the numbers expressed as Person-Months at Risk in corresponding periods in the study group, it is possible to calculate the expected number of irregular discharges in the group under study and treatment. Table IV shows the total number of these to be 9.96 for the full nine-month period. This is then compared to the observed number of two irregular discharges.

The question now is: Is this difference statistically significant? This can be tested by a relatively simple statistical technic involving calculation of the Standard Errors of the numbers involved. When this was done, the observed difference between the two numbers was found to be 2.5 times the standard error of the difference. It is, therefore, believed that the difference between the number of irregular discharges observed and those expected is statistically significant, since it is not likely to be the result of chance alone.

This result is arrived at in the following way:

S = the observed total of irregular discharges in the group studied

ES = the expected total of irregular discharges in the group studied

Let n = the number of person-months in the group

Let $p = \frac{ES}{n}$ = irregular discharge rate/person/month

Standard Error (SE) of $S = \sqrt{npq}$

Standard Error (SE) of $ES = n\sqrt{pq}$

$$p = \frac{9.96}{269.5} = 0.036$$

$$SE\ (S) = \sqrt{(269.5)\ (.036)\ (.964)} = 3.0$$

$$SE\ (ES) = 269.5\ \sqrt{\frac{(.36)\ (.964)}{1{,}644.5}} = 1.2$$

$$SE\ (diff.) = \sqrt{SE^2(S) + SE^2(ES)} = 3.23$$

$$\frac{\text{Difference}}{SE\ (diff.)} = \frac{7.96}{3.23} = 2.5$$

By the same technic it is possible to determine the significance of the difference in number of deaths. This is found to equal 1.8 times the Standard Error of the Difference SE, not quite statistically significant, but nearly so.*

To see if irregular discharges were declining among Negro male patients in adjoining States, five States were queried. Figures were obtained from three of them, all adjoining States with the closest sanitorium about 45 miles (San. A) away. The results are given in Table V.

*Some attempt was made by the State director of admissions to get in patients in fairly good shape. This might account for the decrease in expected number of deaths. This should not influence the matter of irregular discharges favorably for the reverse are more apt to be true, i.e., irregular discharges in this type of case is more likely to occur in those who are physically able to take their own leave of the hospital.

TABLE IV

Comparison of Observed Numbers with Expected Numbers of Irregular Discharges in Study Group

Time Period in Months	NUMBER OF PATIENTS — Present at Beginning of Period (Ox)	Number Withdrawing for Specified Reasons — Died (Dx)	Discharged with Consent (Cx)	Irregular Discharge (Sx)	Close of Study (Wx)	Person-Months at Risk (Lx)	Number of Irregular Discharges Expected in Period (Msx X Lx)
0-1	75	5	1	0	4	70.0	2.03
1-2	65	6	0	1	7	58.0	2.15
2-3	51	2	0	1	8	45.5	1.73
3-6	40	2	1	0	25	78.0	3.20
6-9	12	0	4	0	8	18.0	0.85
TOTAL		15	6	2	52	269.5	9.96

$$Lx - Ox - \frac{(Dx + Cx + Sx + Wx)}{2}$$

Obs. Irreg. Disc. = 2 S.E. (diff.) = 3.23
Exp. Irreg. Disc. = 9.96 Difference = 7.96 = 2.5
Obs. Diff. = 7.96 S.E. (diff.) 3.23

It is seen from Table V that irregular discharges were not declining elsewhere during the period of the study but were actually on the increase.

C. Other Data Secured as a Result of the Study

1. Evaluation of Items of Stress (Table VI).

2. Evaluation of Attitudes.

A subjective evaluation of attitudes in five different categories was made with results as shown in Table VII.

IV. *Discussion*

It is evident that in a disease like tuberculosis a great many factors must be taken into account both with respect to its pathogenesis as well as its treatment. Paradoxical as it may seem, certain contributory factors may be more important than the bacterial cause itself insofar as the production of progressive infection is concerned. Many of these factors make their appearance before clinical disease can be recognized and continue to exert their influence after the diagnosis is made and treatment instituted. Included in the list are particular stresses and strains, a wide variety and number of attitudes determining the degree of personal adjustment, and, of particular importance in the problem of long term hospitalization, the total personality of the patient who must make the necessary adjustments to the situation that tuberculosis brings about. Therefore, the hospital physician who finds himself in charge of a patient suffering from tuberculosis must come to some reasonable understanding of such factors if the patient is to receive the most help that is possible to give.

The study described in this paper afforded the writer an opportunity to consider some of the factors that enter into such an illness as well as to attempt some measures calculated to relieve problems in which such factors played a role. An objective discussion of these matters and how they may be managed, as well as an evaluation of the results, are the principal aims of this section.

In the first place, the main objective of this study was to assist the individual patient to endure the ordeal of hospitalization until he could be safely discharged. In furthering that aim, it seems necessary to have some understanding of the problem that confronts a patient with tuberculosis. Let us first consider, therefore, why a patient enters a hospital for

TABLE V

EXPERIENCE OF OTHER SANATORIA IN THE SOUTH ATLANTIC REGION WITH REGARD TO TYPE OF DISCHARGE AMONG NEGRO TUBERCULOSIS PATIENTS DURING COMPARABLE PERIODS*

San. A: Showing the Proportion of Irregular Discharges Among Male Patients:

Fiscal Year	Number Irregular Discharges	Total Number Live Discharges	Percentage of Irregular Discharges
1947-48	13	110	11.7
1948-49	11	102	10.7
1949-50	20	105	19.1

San B: Showing Irregular Discharges 1948-50 for Both Male and Female Patients:

Year	Number Irregular Discharges	Total Number Live Discharges	Percentage of Irregular Discharges
	MALE		
1948	38	73	52.0
1949	31	82	37.8
1950	47	103	45.6
	FEMALE		
1948	27	66	40.9
1949	19	80	23.7
1950	26	90	28.8
	TOTAL		
1948	65	139	46.7
1949	50	162	30.8
1950	73	193	37.8

San. C: Showing Per Cent of Irregular Discharges, Both Sexes:

Period	Number Irregular Discharges	Total Number Live Discharges	Percentage of Irregular Discharges
6/30/48	28	209	13.4
6/30/50	49	200	24.5

*Reproduced in the form submitted by these sanatoria.

treatment of tuberculosis. What else could ordinarily inspire him than the belief that this is the place to come to get better? Why, then, does he so frequently decide to leave without medical sanction? We might begin the attempt to solve this puzzle by estimating what the patient's feelings must be during this stage of his illness. First, the diagnosis has probably been a shock to him. That this is generally so was confirmed in the present experience as well as that of many others.[1-8] Although the patient arrives still feeling anxious and more or less depressed, he is apt to be hopeful, especially if his case has been handled well by the diagnostician. What he learns and sees, however, after entering the hospital may do nothing to reassure him and, on the contrary, merely intensify his anxiety.

He is lonely and homesick. His fears are increased when he sees the condition of others: the critically ill, those who have been in months and

TABLE VI

EVALUATION OF STRESS

	‡‡		†		§		Total		N.D.
	Per cent of		Per cent of		Per cent of		Per cent of		
	No.	Total	No.	Total	No.	Total	No.	Total	
Economic Stress	16	22.6	27	38.0	28	39.4	71	100.0	4
Social Stress	40	56.4	16	22.6	15	21.0	71	100.0	4
Occupational Stress	38	54.4	12	17.2	20	28.4	70	100.0	5
Alcoholism	20	29.4	10	14.6	38	56.0	68	100.0	7
Other Disease	18	26.0	8	11.6	43	62.4	69	100.0	6

‡‡ Considered by author as very severe.
† Considered by author as moderately severe.
§ Considered by author as insignificant.
N.D. No data (Pt. moribund, in hospital too short a time, etc.).

TABLE VII

EVALUATION OF ATTITUDES IN THE GROUP

	‡‡		†		§		Total		N.D.
	Per cent of		Per cent of		Per cent of		Per cent of		
	No.	Total	No.	Total	No.	Total	No.	Total	
Toward the disease	13	17.8	10	13.7	50	68.5	73	100.0	2
Toward self	16	22.9	13	18.6	41	58.5	70	100.0	5
Toward others	7	10.0	16	23.2	46	66.8	69	100.0	6
Toward the doctor, institution, etc.	9	13.0	10	14.5	50	72.5	69	100.0	6
Toward work and play	6	8.7	10	14.5	53	76.8	69	100.0	6
Main interests	11	16.4	7	10.4	49	73.2	67	100.0	8

‡‡Very unsatisfactory
†Mildly unsatisfactory.
§Satisfactory.
N.D. No data (Pt. moribund, in hospital too short a time, etc.).

years, the many who have had several admissions and perhaps disfiguring operations, and the witnessing of death which is almost a daily affair. He may begin to ask himself: "Does anybody ever get well?" He finds it easy to identify himself with these luckless patients. A loss of hope ensues. Add to this the results of staff indifference plus family problems that he has left behind, and he readily concludes that he is in a hopeless predicament. If this situation develops, the stage is set for early departure.

Obviously the physician should be aware of the possibilities inherent in such a situation and prepare to take constructive action. He should look for these difficulties and not be surprised when he finds them. But how does he go about determining what difficulties exist? Certainly not by an authoritarian approach to the matter in which the patient is merely "told" what he must do. Instructions in the facts of his illness should be withheld until the patient is properly conditioned to get maximum benefit from any such instructions. This seems best done by first finding out what the patient thinks about his disease and the general situation in which he now finds himself. This is readily ascertained and allows the physician to estimate more correctly what problems exist and what needs to be done. The discussion technic previously described favors the disclosure of this knowledge and unless something like this is done, the physician may never find out what problems exist, a knowledge of which may have forestalled irregular discharge.

The initial interview is particularly important. The manner in which this is conducted helps set the tone for all subsequent ones. In the work here, the first contact was more in the nature of an informal visit with the patient. Therefore, no attempt was made to make a written record at that time. The writer felt there would be plenty of time during succeeding interviews to get details necessary for record purposes. This is not an unreasonable hope, since we expect to have the patient with us for a considerable period. We are not dealing with a disease like lobar pneumonia in which case the issue is settled in one way or the other within a rather short space of time. Furthermore, for the moment we are in possession of a number of medical facts concerning the patient from the medical advisors who arranged for his admission.

Consequently, it seems more important at this juncture to ascertain what the patient thinks has been going on in his case. Some of the things we

TABLE VIII
PERSONALITY DISORDERS

	Number	Per cent of Total
Marked personality disorder	19	27.5
Mild personality disorder	10	14.5
Insignificant or none	40	58.0
TOTAL	69	100.0
No data	6	

want to know in this connection are: what he has been led to conclude about his disease, his treatment, and expectations for recovery. These are some of the reasons for making the interviews patient-centered.

In this approach the physician looks for cues in the patient's statements or questions about such matters in order to decide where to begin with the patient. Thus, the physician can start "tuned in," as it were, with the patient at the point where the latter now is in his thinking. For example, the patient may make some remark about his x-ray films or perhaps the tuberculin test which can then provide the cue for a discussion of the diagnosis. Or, he may ask some question like, "Do I have it bad?" which can lead forthwith to a discussion of the patient's case, emphasizing, among other things, its individuality, the respective roles of the patient and the physician in attaining "the cure," length of hospitalization, etc.

In this type of interview, the physician plays the role of "active" listener. That is to say, he has some mental plan based on experience with such patients, concerning the manner in which he shall deal with the problems that commonly arise. But he helps the patient bring them up first. In that way problems are met one at a time and the common error of giving the patient more than he can properly assimilate is avoided.

When a good rapport has been attained and some problems found to work on, the point is reached where other technics can be employed effectively. These technics, such as education and explanation in the facts of his illness, re-education and suggestion in instances where knowledge and attitudes are faulty, and reassurance and support in order to maintain hope in recovery all prove useful toward the general goal of helping the patient adjust satisfactorily to the hospital setting. These are technics which are employed successfully in psychiatric work over a wide range of behavior problems, among which the problem or irregular discharge of the tuberculous is certainly one. They are here called mental hygiene technics in the sense that they are employed in the interests of prevention. Although modern psychiatric practice has made possible more precise knowledge of their usefulness, value and limitations, they were not unknown to physicians of an earlier day. One has only to think of physicians like Osler, Peabody, and many others to recognize in their work similar practices to what is urged here. Taken as such, they constitute a good part of what is known as the "art" of medicine.

The utility of such technics depends in large measure on the knowledge and skill of the physician using them. Fortunately, the skills can be acquired by physicians who, although not psychiatrists, are sufficiently interested in developing them. The knowledge needed is concerned with the disease itself and an understanding of the individual patient and his problems.

With regard to the first, the physician must obviously be in possession or a rather full understanding of tuberculosis as a disease. This knowledge can then be gradually imparted to the patient in words and concepts which the latter can understand. In doing that, the patient will be led to understand certain things about its cause, how it may be arrested, and what is

necessary for his rehabilitation. He is informed that the average patient requires a full year, more or less, of hospitalization in order to start him on the road to recovery. The author feels it to be a mistake not to tell him this early. Even though it sounds like a long time (and it is), the final effect is more likely to be reassuring rather than the reverse.

In order to minimize the unfavorable reactions that result from seeing other patients do poorly, the individuality of each patient's case is emphasized. No two cases are exactly alike. Recovery depends upon the extent of disease and how well his treatment is conducted, with a good share of the responsibility depending upon him. His attention is directed to the large numbers that get well. In that way he is more apt to keep oriented to recovery and show less tendency to identify with patients not doing well under treatment.

The patient's questions about the facts of his illness and its treatment are discussed with him in a sympathetic manner. Therefore, over the course of the long hospital stay, a sound knowledge of his case is obtained, a knowledge which can be used to advantage in adjusting to his illness, both in the hospital and in the period after discharge.

As an aid in attaining the goal of adjustment, the physician will want to know some details about the patient which may be overlooked if attention is focused only on the medical aspects of treatment. To help do this, the author found it convenient to maintain a standardized form giving simple biographical details about the patient, gathered as he went along in the interview work. Particular attention was given to the discovery of certain stresses that may have acted as contributory factors in the pathogenesis of the illness. Such knowledge can be turned into account in the program of general life adjustment of the patient after he has left the hospital. It also provides some material for speculation as to the probable importance of such factors in the problem of tuberculosis. Therefore, a few remarks about the stresses reported by the patients in this study group do not seem out of order.

To repeat what has been said in the beginning of this discussion, infection with tubercle bacilli is not the whole story in the problem of adult reinfection tuberculosis. There seems to be a definite correlation between a high incidence of tuberculosis and the presence of certain other internal and external factors. Factors frequently mentioned in the literature are genetic constitution, malnutrition, the presence of certain other diseases like diabetes and silicosis, socio-economic conditions and others. It is interesting that in the group under consideration here, only 27.3 per cent (see Section III) gave a clear history of contact with a known infectious case. Varieties of stress, however, were numerous. Social stress and occupational stress were particularly high. In the first named, conditions were very unsatisfactory in over half (56.4 per cent) of the patients. Again over half the patients (54.4 per cent) reported marked occupational overstrain with another 17.2 per cent indicating such overstrain present in a moderate degree. Examples of such overstrain were found in cases where patients carried out another job in addition to the regular one, others working under

conditions demanding heavy work unprotected from the elements, and under high pressure, "step-up" operations over a long period of time. Terris,[9] as a result of his studies, suggests the possibility that the conditions of occupation, especially physical overstrain, may play an important role in tuberculosis.

Other adverse factors were also noted in this group, including economic distress in the majority, excessive alcoholic indulgence in a majority, and in 37.6 per cent the presence of some other disease was reported as antedating the diagnosis of tuberculosis. It is, therefore, plain that a proper understanding of tuberculosis demands a consideration and evaluation of many factors.

As a further step in the effort to understand the problem, an attempt was made to identify and deal with certain factors in the personality of the patients. The biography referred to above is believed to have been useful in helping to formulate some idea of the pattern of personality functioning in each of the patients. A notation of the attitudes expressed and displayed by the patient during the interview work and in the hospital setting was found to be of additional use in formulating this pattern. As a result of this combined analysis, disorders in personality functioning during premorbid life was found in a fair proportion of the group (Table VIII). It is seen there that 19 (27.5 per cent) of the 75 exhibited disorders of marked degree with alcoholism in first place with six, and psychoneurosis in second place with five patients. It is impossible to say what role such disturbances might play in the etiology of the disease. It is not inconceivable, however, that long-standing anxiety, interfering as it does with proper adaptation to the problems of everyday living, may in some instances be a decisive factor in the development of active tuberculosis. With regard to these 19 patients, however, there seems little doubt that the personality disturbances encountered were sufficiently marked to justify some type of psychiatric management.

Although the role of personality in tuberculosis has long been the subject for debate, little is known concerning its true relationship to either the development or the course of the disease. It does seem to be true that a rather large proportion of patients display personality disorders of one sort or another. This appears to be the case in the present study, as well as others that can be mentioned. For example, Day[10] observed evidences of such disorders in 30 per cent of patients in an English tuberculosis hospital, and Forster and Shepard[11] in America found such evidences in 31 per cent of their patients.

At the same time, one must remember that in none of these studies, including this one, was there a corresponding sample of the non-tuberculous population evaluated for personality disorders. It would also appear of some significance that the majority of patients show no such disorders by any of the commonly employed criteria, nor is there any particular disorder shown to be common to all patients in the affected minority. Furthermore, although a history of long-standing anxiety is not uncommon in these patients, anxiety per se is not necessarily a forerunner of tuberculosis. If

that were so, we might expect to find a heavy incidence of the disease in conditions where anxiety is a prominent feature in the clinical picture. Such is not the case, however. In the clinical entity known variously as "Anxiety Neurosis," "Neurocirculatory Aesthenia," as well as by other names, the death rate from the common causes of death, including tuberculosis, appears to be no higher than in the general population. The problem is further complicated by factors of stress which may be of importance in the production of behavior disorders as well as tuberculosis. At least they would seem to merit our attention, since they can be studies with a view to a better elucidation of the relationships involved.

Nevertheless, regardless of any role that personality may play in the pathogenesis, there appears to be valid reasons for a scrutiny of personality in attempting to help patients with the problems of hospitalization. In this study, a particular effort was made to approach the matter through a consideration of certain expressions of the patient's personality functioning, called attitudes. By way of introduction to that aspect, an attitude may be considered to be a state of more or less enduring neural readiness to act in a certain way. Learning and conditioning play important roles in its formation. An attitude is formed in relation to an object, a person or value which may or may not have affective connotations in the beginning, but eventually develops affective properties by the time the attitude is well established. We may take as an example the way an individual forms an attitude towards tuberculosis. Persons living in communities where tuberculosis is present invariably develop some type of attitude toward it, either as a result of their own observation or perhaps more commonly from what they have heard concerning the disease. An individual's experience is frequently such that he comes to variously regard the diagnosis of tuberculosis as the equivalent of a death sentence, or that it implies constitutional weakness, or as some form of punishment. In this way, it may come to be associated with feelings of fear, inferiority, and disgust. Therefore, his reaction on being told that he has tuberculosis depends, to a great degree, upon what attitude he has already developed with regard to it. With the cultural attitude being what it is, the physician should not be surprised that the diagnosis frequently results in a great deal of dismay, anxiety, and even depression in so many patients. Others, as a defense against their feelings, may even be led to deny the diagnosis or its implications. It is easy to see the practical importance of knowing something of the problem of attitudes in the management of patients. It is also well to know that attitudes, although they are relatively enduring states of readiness, are not permanently fixed or immutable, but on the contrary are capable of considerable modification. This means that a faulty attitude that might result in the patient's taking action contrary to his best interests can, through use of the technics discussed here, be modified or replaced by ones making possible good adjustment. Therefore, attitude therapy is an important part of the work in preventing irregular discharges.

Obviously, the number of attitudes a patient can hold are as numerous as the objects, persons, and values represented in real life situations.

Thinking now of just the tuberculosis patient, we know he invariably has some preformed attitude about his disease. We should also know that he has certain attitudes toward the hospital and the staff where he is being treated. Again, such attitudes may have been formed before he ever reached the hospital. From hearing of the experience of others there, he may have decided that the hospital offers little hope for cure; that is to say, it is a place for people to go to die rather than get well.

Again, in the intimate interpersonal setting of the hospital, attitudes formed toward himself and others prove to be of importance. If he is rebellious, suspicious, and quarrelsome, institutional life is apt to prove miserable for himself and the others who must deal with him. As one might expect, irregular discharges are common among those who act in that way. At the other extreme, there is the overly passive and dependent patient who recognizes in the hospital a long sought refuge, with the result that he may resist efforts to discharge him when the staff considers him ready to go. In another direction, attitudes reflected in main interests, goals, and desires may prove to be important factors in the problem of hospital adaptation. A patient who is a parent,, let us say, with interests primarily centered in his family, will present different problems from that of the unattached person.

Many other examples of the way attitudes can affect the outcome could be cited were space sufficient. Suffice it to say that the physician should become sensitized to the existence of these aspects of personality functioning, since a knowledge of a patient's attitudes yield a useful clue to the patient's personality, without which knowledge the physician may find himself working in the dark in tackling the problems of hospital adjustment. One way of increasing the staff physician's sensitivity to such aspects of individual functioning might be through the maintenance of staff contact with a psychiatric consultant able to provide staff orientation in common psychiatric practices.

In the foregoing pages, the author has attempted to describe certain aspects of the problem in the prevention of irregular discharges among tuberculosis patients. The observations and procedures depicted there are, in the nature of the effort, not measurable in exact terms and, at best, they would seem to correspond more to principles expounded in describing an art. Thus, it seems to be somewhat like the rules a painter would give in describing how he goes about painting a portrait or a landscape. The results of the experiment, however, are susceptible to measurement and these shall be discussed briefly at this point.

In the first place, the results are of a type that can be accurately depicted, i.e., the patient either dies or he is discharged alive in either of the two ways set forth here. An important step in proceeding to an evaluation was to have a group available which was, in all essential respects, like the group in which the experiment was conducted, except that mental hygiene technics were not deliberately employed in an attempt to influence the type of discharge. In taking the group described in Section III for comparison, a number of factors had to be taken into account in order to

assure comparability. If we can trust the analysis favoring such comparability, it is then possible to employ the methodology of a modified life table technic in evaluating the efficacy of the special efforts.

The life table technic has been well established as a reliable method of measuring the experience in a group where individuals enter and leave at different times during the course of observation. Frost[12] was among the first to utilize this technic in the study of public health problems, and later on his collaborators and students did much to extend and broaden its scope. In a recent editorial, Sartwell[13] has called attention to the possibilities of utilizing a modified life table technic over a broader range of treatment problems, especially in the chronic diseases where concurrent controls are difficult or impractical to establish. The modified life table technic employed here is in line with such recommendations. It was found here, for example, that one could make use of the experience of the earlier group admitted over a two-year period in determining the probabilities of occurrence of a particular event, e.g., irregular discharge in the population exposed to risk. The use of the concept person-time unit of observation with the time unit considered as one month was employed in this undertaking. The fundamental data required for analysis was readily ascertainable from extant records, making it possible to obtain the data of entry of the group exposed to risk, and the data when a particular event transpired.

It would seem that this method offers obvious possibilities in the study of mental health problems. Siemens[14] has employed a version of the life table technic in a study of infant development centered about the problem of weaning. Adequately refined diagnosis in the behavior disorders should be able to yield the type of data that could lend itself to comparable manipulation where it is desired to evaluate treatment.

V. *Conclusions*

A. Irregular discharge of patients at the institution studied was found to involve a substantial majority of those discharged alive. Most of these left during the early weeks and months of hospitalization there, indicating the need for special efforts during the early period of hospitalization.

B. A method of procedure using the simplified mental hygiene technics of discussion, explanation, reassurance, suggestion, education, and re-education during the course of the patient-centered interviews was developed in an attempt to prevent many of these irregular discharges. This method lies well within the capacities of the normally competent chest physician with perhaps some staff guidance from a psychiatric consultant. Comparatively little time was required in this study, averaging 1.3 hours per patient per month. The work with the patient proceeds with the gradual accumulation of data regarding life history, estimation of stresses, and determination of attitudes.

C. Application of these methods revealed that:

1) Stresses of various sorts were prominent features of their premorbid life. Occupational stress was most outstanding and may help to account for the significantly higher death rate of the male Negro.

2) Unsatisfactory attitudes were found in many cases but did not loom as large as other factors of stress.

3) No specific personality type more commonly encountered than any other was discerned by the methods pursued in this study.

4) Deaths were less than expected which may be accounted for by a changed admission policy.

5) A statistically significant trend occurred in the direction of a decrease in irregular discharges, accompanied by a rise in the number of discharges with consent.

6) Mental hygiene efforts similar to those used here should be included in the treatment of tuberculosis patients.

D. A new application of the life table technic was developed which can be applied in the statistical evaluation of outcome whether it be in terms of irregular discharge, discharge with consent, or deaths.

SUMMARY

Factors related to stress and psychological functioning in the patient with tuberculosis are reviewed and considered. Irregular discharges as an important problem in the hospitalization of such patients was significantly influenced in a group of 75 adult male patients by the application of simple mental hygiene technics. A new form of modified life table technic was devised for use in evaluating the results of this study.

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RESUMEN

Se revisan y se consideran los factores en relación con el esfuerzo y la función psicológica en el enfermo de tuberculosis. Las altas irregulares de hospital son influidas de manera significativa por la aplicación de simples medidas de higiene mental en un grupo de 75 varones adultos enfermos.

Se ha ideado una nueva forma de tabla vital para valuación de los resultados de este estudio.

RESUME

L'auteur fait la revue des différents facteurs concernant la psychologie des tuberculeux. Les départs non motivés de tuberculeux hospitalisés furent nettement influencés chez 75 malades du sexe masculin par l'application

des simples principes d'hygiène mentale. Une nouvelle méthode d'investigation à été établie pour évaluer les résultats de cette étude.

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Group Therapy in a County Tuberculosis Sanatorium*

A Psychologic Approach to the Problem of the Chronically Ill Confined Patient

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Tuberculosis patients in a sanatorium have been seen in groups at weekly intervals in an attempt to help them adjust to the emotional problems of their illness. This is a two-and-a-half-year study of *group orientation therapy* with organically ill non-psychiatric patients. This problem is of primary practical interest to practitioners of medicine.

Group psychotherapy has become a useful tool of the psychiatrist. Its potentialities were clearly demonstrated during the last war, but have not been evaluated in the case of medical illness. The first group therapy of any kind was carried out by Joseph Pratt[1] in Boston, with non-hospitalized tuberculosis patients, forty-seven years ago. Since his papers, I have found no published work on this subject.

The tuberculosis sanatorium offers a unique setting for group therapy, for the patients' problems of adjustment are similar, predictable and involve common social, medical and personal situations.[2]

The Problems in General Terms

We are concerned with three factors: (1) the sanatorium; (2) the disease, and (3) the person. Each sanatorium has a culture of its own, but in general has features similar to other sanatoria. However, patients' attitudes differ, depending upon their life experience and their cultural background.[3,4]

A large number of patients leave sanatoria against medical advice. From published studies in this country[5] it appears that close to one-third of the patients leave against advice and close to one-half of these have moderately far or far advanced disease, with positive sputum. The practicing physician is, therefore, concerned with the fact that in a third of the instances the sanatorium does not satisfactorily meet the purpose for which he sent the patient to the hospital. Rarely do patients leave against advice because they are dissatisfied with medical treatment. The reasons for leaving can generally be attributed to emotional problems with their core in personal and economic factors. The patients who do not go AWOL must still handle the feelings which motivate the others to leave.

An unknown number of patients probably suffer longer or become sanatorium- or cure-invalids, and their disease may be poorly controlled by

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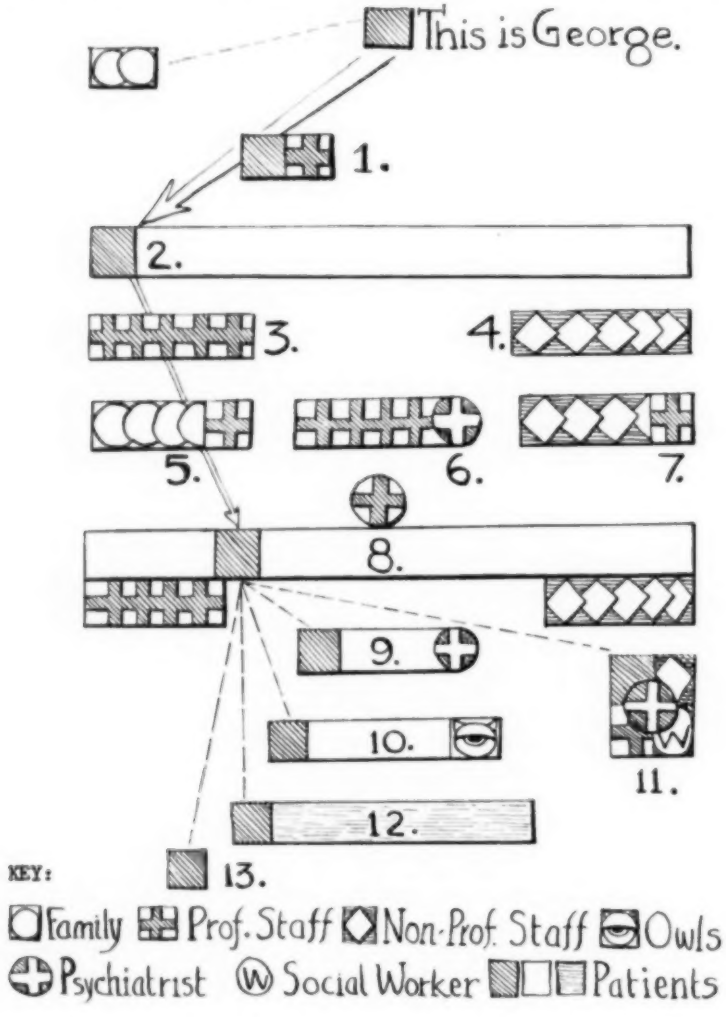
fluctuating native and acquired resistance. Numerous isolated cases of precipitation or exacerbation of tuberculosis in the currents of rage, anger, resentment and hostility have been reported. No purpose will be served by citing any of these striking cases. This remains, however, to be the subject of a carefully controlled scientific study. We can only regret and be amazed at the fact that almost all these reports have been formulated in the hazy setting of opinion and memory.

Method and Setting of Study

San Mateo County Tuberculosis Sanatorium is a hospital of 105 beds, located in a valley in the country, drawing upon a population of better



than average financial and intellectual resources, and including a minority population of Mexican, South American, Chinese and Negro patients. It is a well run hospital, lacking nothing in physical equipment.

Group work may involve the following groups:

1) Patients
2) Professional staff
3) Non-professional staff
4) Entire hospital mobile population
5) Family and visitors
6) Inside-Out Club
7) About-Face Group (Owls—AWOL).

From the patient population a group of eight patients of mixed sex and nationality was selected and seen at weekly intervals for 45 minutes, over a three-month period. Other groups are seen at monthly intervals: visitors who are willing to listen to a brief talk and participate in discussion after visiting hours; the professional staff; the non-professional staff, and the entire hospital population of patients who were able to come to a common room, together with the professional and non-professional staff. This latter group consisted of about 50 people.

Individual patients were seen at their own request or the request of the staff, but this was not frequent.

Two additional groups are contemplated: the first, which I call Owls, was conceived to work with so-called "recalcitrant" patients leaving or threatening to leave AWOL. The reason this group was never in operation was that the need never arose after the operation of the other groups. The idea of this group was that members should conduct it themselves, in a way vaguely similar to Alcoholics Anonymous. This begins with the premise that they may accomplish most through their own efforts, and that people suffering from emotional conflicts of this sort speak the same language and can understand one another; that these patients are emotionally ill.

The last group contemplated is a social club where recently discharged

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FIGURE 1 illustrates diagrammatically the way in which the present scheme touches the patient's life. For the sake of narrative the patient will be called George. He is indicated by a cross-hatched square at the upper right hand side. He is separated from his family, symbolized at upper left by incomplete circles. He enters the sanatorium at Point 1, where he is met by a member of the professional staff (the sooner the better—ideally, within an hour). George now becomes a member of the sanatorium population, indicated by the long white bar at 2. The crosses at 3 indicate the professional staff, and the diamonds at 4 the non-professional staff: aides, orderlies, etc.

Three separate group meetings help set the background for patient groups. These are: at Point 5, meetings with family and other visitors, led by a professional staff member; at 6 the professional staff meets with the psychiatrist, and at 7 the non-professional staff meets with a member of the professional staff, preferably the superintendent. At Point 8 the entire hospital population able to gather in a single room meets with the professional and non-professional staff, led by the psychiatrist. At Point 9 George meets in small group therapy. At Point 10, if George is in danger of going AWOL he may become a member of the Owls. At Point 11, any person in the hospital may meet individually with the psychiatrist. No intensive psychotherapy is undertaken. At Point 12, after discharge, the patient may participate in the Inside-Out Club. At Point 13 George is discharged and on his own. In his course through the sanatorium his only contact with the psychiatrist may be in the large group at Point 8, or at 9, 10, 11 or 12.

patients may meet once fortnightly in a nearby town, in a social and educational setting, with the presence of one of the hospital personnel (Figure 1).

We are handicapped by the fact that there is no social worker at the Sanatorium and only two staff doctors. Economic and practical family factors are so important that unless they are adequately aided by social services some anxieties can never be relieved by any psychotherapy. The services of a good social worker are essential to every sanatorium and a psychiatric social worker will probably some day be acknowledged as equally important. To realize the full value of such a program as outlined, it must operate continuously and must have the active participation of more than one person interested in such a venture.

Although the number of patients who signed their own release with the approval of the staff continued about the same during the active group therapy work, no patient went AWOL and no patient was committed under the Tuberculosis Quarantine Law during the time that the patient program was in full operation (10 months). In the three years preceding, eight patients left AWOL, and a similar number with positive sputum was held under quarantine. It was necessary temporarily to discontinue the group work, and in the following month one patient was quarantined. One cannot discount the element of coincidence, neither can one discount, at this time, a casual relationship. From personal observation, from interviews with patients and comments of staff, from questionnaires and letters it can be inferred that morale increased and that some patients received quite apparent help. Similarly, certain changes occurred in hospital routine, which led to the more happy adjustment of patients to their difficulties. This could not have occurred without a cooperative and interested staff. No obstacle at any point was encountered by the psychiatrist.

Goal and Limitations

For practical purposes and for the sake of brevity, the small patient-group will be mainly dealt with. No effort is made to "treat" individual patients in the sense of making major alterations in individual neuroses. In fact, the patients with crippling emotional conflicts are not good candidates for the small group. The aim at every turn is the amelioration of anxiety and depression, the facilitation of understanding of everyday practical problems of sanatorium life for the "average" patient.

Both the therapist and the patients change to some degree. The change operates almost entirely from the interaction of people free to talk without fear of restraint, censorship or punishment. Patients obtain encouragement, insight and help from one another more than from the therapist. Feelings previously bundled up in self-centered interest are now directed outward. One patient may, for example, vigorously attack a doctor or a nurse, while another pleads their case. Sides are often taken but group decorum must never be permitted to reach peaks of undue excitement. The patients help one another with conscious material. Interpretations are cautious and gentle.

Group Selection

The group is random and artificial and is not a substitute for a family or any customary social unit where gratification is obtained, but feelings in relationship to these other units affect group feeling. Groups are chosen in consultation with the Medical Superintendent, to include patients in the sanatorium from three weeks to many months or years. Patients who would be anticipated to monopolize group sessions unduly were excluded; they can be dealt with in separate groups. Generally we have attempted to obtain a sample cross section of the sanatorium population.

Patients should not be in these groups during the first three weeks of their hospital stay, for in this period they do not easily take to the criticism of the sanatorium and of one another that flows so freely. There is at first a strong need to believe that however "bad" this situation seems to them, they have not made a mistake in coming to the sanatorium. They should be left to their own devices to meet the initial acceptance, with individual help as needed from their own physician. As group work proceeds it is generally found that patients bring friends and roommates into the group without asking permission. This is permitted by simply acknowledging it. Often these people become permanent members of the group, or come only once or twice. Groups tend to swell in members by their own selection, and to sift by a natural process of accretion and shedding.

Various other methods of selection have been experimented with, but it was found that it made little difference how the groups were selected, for they soon became self-selecting and all seemed equally interesting. Who is chosen is not nearly so important as how groups are conducted, and this varies with the group by its own choice. No difficulty was encountered in racial mixtures, and indeed, interesting material was stimulated and handled in this area. The mixture of sexes was felt to be highly desirable; it tended to add an occasional note of chivalry and a healthy moderating influence, and in addition, it increased interest and attendance.

For every one who dropped out, more came or wanted to come. The most common reason given for dropping out (from personal interview and questionnaire) was that the "same old thing" was always discussed. On going back over notes on meetings it was found that a great variety of material had been introduced, but at each session the person who subsequently left the group had usually contributed the same idea and had not heard what the others had to say. No effort was made to entice people to come or return, for it was felt that group work of necessity is suitable only to a portion of a hospital population, and attendance was strictly voluntary.

It was found that there developed in the sanatorium a prestige value among certain types of patient for being members of a group. The patients seemed to be conscious of belongers and non-belongers. They were sometimes reluctant to share or talk about group activities to non-members, often in fear that they would be ridiculed for attending. Since groups were necessarily small, it was decided that there would be some merit in bringing into certain group activity as many patients as possible. This was accom-

plished by two devices. First, the psychiatrist met with all newly admitted patients as soon as a group of at least eight new patients had accumulated. They met once only and were given a formal lecture concerning the emotional problems of adjusting to tuberculosis. This also served the additional purpose of permitting new patients to see the psychiatrist and realize the friendly setting of a group. Attention was usually keen. The second device was the establishment of the large hospital population group. Material brought into this large group was strikingly different from that introduced in the more intimate small groups. Complaints and suggestions about sanatorium housekeeping, food and personal gripes were more uncritically aired in the safe, large group, and there was a more artificial atmosphere. It was a fortunate coincidence that small groups were already in operation before the large group was undertaken, for members of the small groups often bridged awkward moments and they were less selfconscious.

Difficulty in interpreting the value of the large group is almost insurmountable, and its value is equivocal. If it met often it was almost solely repetitious, and the patients who braved the entire hospital population with a major complaint usually ceased attending subsequent large group meetings. Although they usually accomplished at least part of what they set out to do, they tended to feel stranded and unsupported when all the patients did not rise up in a chorus of collaborative indignation. They were often amazed to find divergent opinions about such simple things as how clean the silver was or whether the soup was palatable. By and large, the medical and non-medical staff did a fine job of answering and satisfying patients in a mature fashion. The gentle support given both sides by the moderator-psychiatrist often left both factions feeling slightly betrayed. Patients did more cavilling without being brought to account, as in the small group. Nonetheless, large group activity and sanatorium repercussions led to lively analysis in the small group meeting the following week, where the patients were less dogmatic and more friendly.

Therapist's Role and Attitude

The therapist must be careful not to stir up anxieties, must be willing to follow the most unconventional avenues where the group will lead him. He must be clearly aware that he is dealing with people who generally do *not* feel they need psychotherapy, and must set the atmosphere in the comfortable feeling that the group is, after all, only concerned with everyday human needs and that he does not consider the patients as subjects for study or objects of therapy. This factor sets the psychotherapist with organically ill patients apart from psychotherapy with psychiatric patients. Yet, like psychiatric hospitalized patients, the group must handle feelings similar in certain respects—for example, What will you say to an outsider, after discharge, who says: "Oh, you left the bug house?" (stigma) and "Do you tell them about your disease and will it interfere with employment?" (fear of reprisal). These feelings occupy a considerable degree of patient interest. They also will externalize their feelings about their doctor, sometimes with shattering frankness, as: "I wish my doctor (or

you) would drop dead." They will often bring up their own problems cloaked as an absent friend's plight.

The therapist must never outrage a patient and must always abide by the rule that if he can't do any good he must not do any harm. It should be kept in mind that behind this particular group therapy concept is the limited duration of group activation. It is planned that any individual group be in existence only for twelve to fifteen sessions, so as to reach as many patients as possible and to deal with problems with reasonable dispatch.

The old proverb that there is more than one way to skin a mule applies to the therapist's attitude and role. To try to define the qualifications of an ideal leader for the sanatorium population would be impossible at this time. However, it is probably true that the personality and attitude of the leader are of greater moment than his methods. Whether or not he has had tuberculosis is of considerable importance to himself and to the group.[6] He must be prepared to handle this point with sympathy and understanding, and while he may judiciously draw upon his own experience or memory, he must avoid obvious interjection of his personal opinions and personal solutions. He can neither be an oracle nor rely upon his own experience with the disease to maintain an authoritarian role, which many patients will seek to establish or demolish. He is an authority sine authority.

Having been a tuberculosis patient myself I find that on a few occasions reference to it by me or the patients may be of considerable help. There is an obvious bond, but there are mixed feelings, for they often alternately consider me a partisan of theirs and of the staff. The patients will realize, without reciting the fact, that here is a man who has overcome the disease and is carrying on in the normal stream of life. For some, this fact will mobilize resentment and envy; for others, admiration or inspiration; for a few, indifference. As one of my patients commented: "After all, everybody has the damn thing."

On occasion the moderator must take an active and decisive hand. If he finds a patient opening the lid on personal problems which can never be adequately handled in this group, he must gently close it. If anxiety reaches a pitch disturbing to other patients, he must truly moderate. He must try to keep a comfortable flow of words, but not feel uncomfortable in occasional periods of silence. He must short-circuit attempts to use the group to lead crusades, which is not its purpose.

The therapist must be prepared to handle feelings within himself about the group and its members, and their feelings about him and one another. While I feel it is by no means necessary that the moderator be a psychiatrist or psychologist, or psychiatric social worker or psychiatric nurse, such training has some clearcut advantages. If the leader has not had tuberculosis, he must not only be alert to the psychologic factors continually unfolding before him but to the medical, pathologic and therapeutic aspects of the disease. He will be a sitting duck at a crucial point if he hopes to learn this from the group or "as time goes on." If the moderator is not a psychiatrist it is equally imperative that he know basic features of psy-

chologic medicine and to have acquired by first-hand experience and training skill in working with patients on this level. He must be prepared to take abuse and control his feelings, for if he does not, the group will not.

Much is accomplished by individuals taking clues from the obvious words, attitudes and maturity or healthy sophistication of the moderator. It is for this reason that his personality is of paramount importance. Within limits, the less he says the better off the group will be, for its members will be forced to work out their own feelings and opinions. The therapist must be content if these are at variance with his, and he must clearly and pointedly indicate that he does not have the answers to their problems but that they do. Nevertheless, he must not permit obviously destructive attitudes and totally ignorant conclusions to take hold of the group. He must correct misconception if it is left unchallenged. This can be done by framing general questions or by a sort of out-loud wondering how they come to such conclusion, pointing out that unexpressed feelings seem to lie behind inconsistent attitudes. Often a quizzical look, a pause and slow restatement of patients' words will strike open doubts in the mind of the individual patient, or if not penetrating his shell, will serve notice to and alert the others to a searching attitude.

He must understand strong liking or dislike of himself and not take pleasure when he has expressed an opinion and is seconded by a group member. When I once indicated that a patient was dealing with highly irrelevant material, another patient attempted to silence the first by saying, "You heard what the doctor said, shut up!" Now he and the group must deal with the feelings of the second patient.

He must avoid reassurances when he is not absolutely sure of them. His attitude must imply the feeling that the patient is secure without dwelling upon the subject. At every turn he must be warm, human and empathetic, never sentimental or sympathetic in the usual sense, nor too kindly. His force stems from the attributes which the group invests in him, not from those he wishes to profess or demand.

The words of the therapist, being few, must be well chosen. His tone of voice, ease of manner and speech must be natural, but on some occasions expressed in such fashion as to produce a desired effect. It is preferable to summarize what has been said from time to time, and usually to avoid participation even if baited. The moderator must be alert for things brought up in a fleeting moment and passed over too hastily, like the fisherman who must harpoon the porpoise above the water, for it threads it way most of the time beneath the surface.

The therapist is responsible for the confidential nature of his relationship. The moderator must, therefore, not divulge material related in real or quasi-confidence. The rest of the staff has no special right to know what transpires in group discussion, but in general terms may be acquainted with it. Notes are confidential and should not be part of general hospital files which pass through numerous hands and are read by many casual, often too-eager eyes. Only after group work has been in existence some time will the general feeling of trust by the patients and the staff permeate

the sanatorium culture. When the patients find that in reality their secrets, gripes and rebellions are respected, will an era of greater comfort exist. Similarly, only after the staff members have themselves come to realize that the group is not a threat to them personally nor to their authority will their confidence be sustained.

Problems Discussed

In general it can be said that patients deal with three great categories of feelings: (1) adjustment upon entry into a sanatorium and to the authority it represents; (2) adjustment to the existing hospital society, the other patients and the visitors; (3) his hopes and longings for an end to the hospital stay and his fear and apprehensions about adjusting to the outside society, to which he must return a seemingly different person. On each level the patient is concerned with acculturation to his illness in changing cultures. The illness and the sanatorium are at once distinct yet inseparable for hospitalized patients. Groups of non-hospitalized patients with whom Dr. Pratt worked several decades ago present a different problem, and in this area I have no experience.

Verbatim Group Discussion

Notes, if taken, should be sparing. Attempts to record sessions during operation lead to stilted and suspicious reactions. Copious notes should be made immediately after the session. In some groups which as time went on became quite at ease, I have taken notes, a sample of which follows. The names, of course, are fictitious:

Bill: My parents are in — — (far away). When I first came down with tuberculosis the upshot was, what should I tell my parents. I decided not to tell them until I was getting well. Someone here said, "Isn't it possible to be an oversolicitous child, like an oversolicitous parent?' I'm protecting them because they are old and wouldn't understand; they'd get excited and worry. I don't think I'm being oversolicitous.

Dick: It could happen.

Mary: I had the same problem. I feel better now that I told them.

Bill: My own feeling is that I'd like to tell them. I hate to keep it up

Tom: Someone else may tell them.

Bill: They do have other sources of information

Dick: You see this all the time, people running their lives for their own sakes.

Bill: Is it being oversolicitous keeping bad news from them? Dad was deeply hurt when my brother was sent overseas. Mother gets excited. To give them two jolts ! Because they are those sorts of persons, particularly because they don't known anything about the disease, better soften the blow.

Dick: Another shock when mother says, 'Why didn't you tell us?'

And so it went. Several days later the patient wrote his parents about his illness.

Another but totally different phenomenon is dealt with in the following discussion, which took place a week after the dramatic appearance of photographs in *Life* of jubilant "cured" patients, recipients of INH (isonicotinic acid hydrazide) and glowing newspaper stories of the new cure:

Bob: It makes no difference to me, there've been a dozen cures since I came into the San. Ask doc. Just another.

Phil: But have they been publicized as much as this one?

Hank: The first night I saw this they showed the headlines on TV. I said, 'I'll bet there'll be quite a reaction.' (In the other patients, unsaid). The key to the thing is that this is a premature announcement. They won't put out public announcements like that in medical papers until it is used a long time.

Betty: I feel elated. Someday (sic) they'll get a real cure.

Phil: Maybe this is the one.

Bob: This announcement makes no difference to me.

Hank: I've seen this in cancer hospitals where I worked, when I heard it I said, 'Take it with a grain of salt.'

Bob: Doesn't make any difference to me.

Phil: It should.

This continued in the same vein. Patients were not demanding the new drug, yet several had asked their doctor, especially the new patients. The group seemed to feel the greatest anxiety and furor would be evident in their families and friends. Their reaction was that they seemed to have been bedded down for a long pull, with the therapeutic armamentarium now at hand. Their optimism was for future research.

An interesting observation is that these patients are usually reluctant to realize that it is "the squeaky wheel that gets the grease." The squeak may not be loud but calls attention to a need. Again and again they point to justifiable and unjustifiable requests, complaints and wishes to talk personally to their doctor, which they do not press upon the staff. These are mentioned to other patients or to a floor nurse, and then stop. It is usually due to (correct or incorrect) feelings that they will produce hard feelings or suffer subtle punishment by going over the heads in the chain of command. If nothing else was accomplished, the patients in these groups began to make direct communication with their doctors about personal problems, and their doctors were receptive.

Success of Group Therapy

Success of this project can be judged in two ways. First and most apparent is continuing attendance and interest in group meetings and noticeable change in individual attitudes. Second, the effect on hospital morale, routines and decrease in numbers of rebellious patients. This is more difficult to assay than the first. Much more elusive is the possible beneficial effect that relief of anxiety and depression has on tuberculous lesions themselves, though changes in appetite and sleep patterns and rest can be studied. Only long-range, carefully controlled studies can answer this important point.

Our project is a pilot study to see if group work is indeed a reasonable and justifiable approach, and what directions it can take. Some psychologists who have recently tried group work in tuberculosis sanatoria have complained of gross indifference of patients. An important feature often neglected is the introduction of novel material of interest for its inherent value, however close or remote it seems to the psychologic problems. The patients themselves give clues to their desire to be given something each time.

The first group, for example, spent many hours developing a story which

was a dialogue between a hypothetical patient and a doctor. Subsequently, with the patients' help this was recorded as a ballad, called *This Is Your World*.[7] From this start various stimulus material was developed. If possible, the moderator should participate in such creative enterprise.

These records which are used with our groups are available to others interested in evaluating this stimulus material. There are three 12-inch records entitled *You and Your Visitors; You and the Sanatorium; You and the Outside World*,[7] and three 12-inch records entitled *Parent Child Conflicts*,[7] short dialogues between parents and children.

Diversity is a keynote, for groups tire of only recorded material. The object being to catch the eye as well as the ear, psychiatric movies were shown, then any good short movie, from popular science to travelogues, and always the patients brought discussion around to the film's meaning in relation to their illness or personal feelings. It seemed to matter little what was shown; the entertainment value *per se* was not considered by the group at large as the primary object. The tone of discussion was set by very few words from the moderator at the end of the movie. Drawings, lantern slides and stories were also utilized. It seems a fair means, for the life of the tuberculosis patient is one of tedious monotony and a feeling of aloneness.

In this sanatorium I am a voluntary consultant, with no financial relationship, status or rank in jeopardy in the professional organization. This has some salutary aspects, for these patients, supported at tax expense, keep a weather eye out for what they might think of as extravagance or financial philandering. Every group in this sanatorium repeats the myth that their doctors have a bean farm, which accounts for the frequency of the item on their menus. In every group there is a degree of outward relief when they inquire and learn that I am not being paid for my services. In a private sanatorium where patients pay for their care and keep, this would have different meaning.

My voluntary status also has an effect upon some patients, in that "if it doesn't cost anything it can't be worth anything," or that my ulterior motives are self-aggrandizement or guinea pig research. For obvious reasons, if such feelings are strong these patients usually drop out of sessions, but, interestingly enough, usually return for one or two sessions later on, perhaps to keep their guardian eye out for what goes on.

There is an element of suspicion about psychiatry, which to many people implies black magic and conjures up thoughts of sorcery. This is not totally removed in the group work, and no effort is made to do so. It soon becomes a relatively unimportant factor as the majority of patients realize that this is a friendly person who wants to help them and not seduce them into a psychoanalysis. Spontaneous expressions of gratitude are frequent and have the same value and meaning as when such similar expressions are made to the cook for a good meal.

Discussion

Group therapy should not be undertaken without judicious forethought.[7] Harm can be done or little accomplished, if it is undertaken without a

clear concept as to its purposes. It seems that the patients' major conflicts involve affective difficulties, those of feelings, usually depression and anxiety. This is further borne out in carefully controlled studies reported elsewhere.[8] Though the group sessions are frequently punctuated by laughter, there is no euphoria or optimism due to the patients' tuberculosis. It is possible that viewing the sanatorium as a therapeutic community, strides will be made toward its change into a more maturely sophisticated society.

Tuberculosis itself has ceased to be the white plague, and great masses of people flock for free chest x-ray films throughout the country. The death rate has fallen from 153.8 per 100,000 in 1910 to 19.6 per 100,000 in 1951, in the United States. Yet despite this, a few voices have been raised, proposing locked wards, and court prison commitments for "recalcitrant" patients has become a matter to be dealt with.

At a time when our country is concerned on every level with more freedom and not less, and the preservation of those freedoms which we have, is it not significant that we hear proposals for prisons in the tuberculosis hospital? I do not doubt that it is wise for a few patients with major psychological problems and illnesses to be confined in hospitals dealing with mental illness or specifically designated for these tuberculosis patients, but that prison wards should become *a part of* the therapeutic community is, to this writer, disturbing, for this threatens the cherished doctor-patient relationship. It echoes Samuel Butler's 1872 novel of Utopia, *Erewhon*[10] where tuberculosis was a crime punishable by imprisonment. How many patients will confide in their physicians, who in the true sense are ministers to the emotionally distraught, when in the physician's other hand is the symbol of his wardenship? Is it not an anachronism that as the danger of tuberculosis to the commonwealth decreases, the drastic remedy should be proposed by a few voices? The element of punishment for a vaguely definable group of "recalcitrant" patients must be handled with great circumspection and caution, and we must analyze most carefully any attempt in the name of illness to take upon ourselves the extra-medical and legal problems of confinement. One cannot help but acknowledge the presence of this problem, now visible on a stage previously cluttered by the monstrous problem of human illness and misery, when one hears frequently the cheer that the eradication of tuberculosis is at hand! But this is no consolation to the patient within the sanatorium who is daily faced with emotional problems.

SUMMARY

Group therapy is proposed as a means of helping hospitalized chronically ill patients with enduring physical handicaps. For two and a half years tuberculosis patients have been seen in group psychotherapy. Small groups of eight to 15 patients were seen at weekly intervals for 12 to 15 sessions. Groups were composed of mixed sex and race. Factors dealing with problems of emotional adjustment and acculturation were dealt with in the group.

Additional group work was carried out in professional and non-professional staff meetings, with family and visitor groups and also the entire sanatorium population which was ambulatory. Group attendance and interest was good.

There seemed to be an increase in hospital morale, amelioration of individual patients' anxiety and depression and a decrease in number of patients with positive sputum leaving AWOL. Certain changes in hospital routines and staff attitudes have occurred.

The group leader should be a psychiatrist or specially trained professional person.

A necessary prerequisite for successful group therapy is an interested and relatively non-rigid hospital administration, willing to adopt a scientific attitude.

It is possible by group therapy to reach a large number of patients with relatively small expenditure of time and effort. The goals of this type of group therapy are not to cure patients of their neuroses nor make them happy, overly complacent patients, but to help them adjust to the necessary ordeal of sanatorium confinement. It is a technique with definite limitations and advantages, and will not appeal to all patients.

Patients themselves, more than the therapist, help one another in the groups. Realistic acceptance of membership in a small group permits easier acceptance of membership in the large hospital group, and possibly a more mature acceptance of the disease and its meaning to the patients' family and friends as well.

This is a preliminary report and no significant claims are made. Further study in several different sanatoria should be undertaken.

The conceptual program could be utilized with other medically ill non-psychiatric patients confined in hospitals.

RESUMEN

Se propone el tratamiento en grupo como un medio de ayuda a los hospitalizados cronicamente enfermos con invalidez física duradera. Por 2½ años se han observado enfermos en grupos de psicoterapia. Se vieron grupos pequeños de ocho a 15 enfermos con intervalos semanarios, por 12 a 15 sesiones. Los grupos estaban compuestos por personas mezcladas en lo referente a sexo y a la raza. En el grupo se trataron los factores concernientes a los problemas de la adaptación emocional y cultural.

En las reuniones de personal también se han estudiado grupos incluyendo en estas reuniones grupos profesionales y no profesionales, con los grupos familiares y de visitantes así como toda la población del sanatorio que es ambulatoria. El interés y la asistencia a las reuniones fueron buenos.

Parece que hay un aumento de la moral en el hospital, mejoría del estado de ansiedad y de la depresión y disminución del número de enfermos con esputo positivo que abandonaron la institución.

Han ocurrido ciertos cambios en la rutina y en la actitud del personal.

El conductor del grupo debe ser un psiquiatra o una persona especialmente preparada profesionalmente.

Un requisito para obtener un resultado satisfactorio es que haya una administración de hospital no rigida, interesada en el plan y deseosa de adoptar una actitud científica.

Es posible por el tratamiento en grupo alcanzar a beneficiar a un gran número de enfermos con relativamente poco gasto de tiempo y de esfuerzo.

El objetivo de este tipo de grupos no es curar a los enfermos de sus neurosis sino ayudarlos a adaptarse a los necesarios cambios que exige la confinación sanatorial, sin pretender extremar la complacencia. Es una técnica con limitaciones definidas y con ventajas y no ha de atraer a todos los enfermos.

Los enfermos mismos, mas que el terapeuta, se ayudan uno a otro en los grupos. La aceptación realista de ser miembro de un pequeño grupo, permite la mas facil aceptación de ingresar en el grupo mayor del hospital y posiblemente una aceptación mas madura de la enfermedad y su significación para la familia de los enfermos y para sus amigos.

Este es un informe preliminar y no se pretende nada significativo como conclusión sino que debe emprenderse mas estudios ulteriormente semejantes a este en varios sanatorios.

Este plan podria extenderse a otros enfermos que no tengan problema psiquiátrico y que esté confinados en otros hospitales.

RESUME

L'auteur propose un traitement par groupe pour aider les malades hospitalisés pour affections chroniques. Pendant deux ans et demi, des tuberculeux ont été suivis dans un groupe de psychothérapie. De petits groupes de 8 à 15 malades subirent des examens hebdomadaires répétés 12 à 15 fois. Ces groupes étaient composés sans tenir compte ni du sexe ni de la race. A l'intérieur de chaque groupe, on etablissait un programme d'accords affectifs et culturels.

Un groupe de travail supplémentaire à été amené à faire des réunions de cadres professionnels et non professionnels, avec des groupes familiaux et des groupes de visiteurs ainsi qu'avec l'ensemble de la population du sanatorium qui était une population ambulatoire. Les résultats obtenus par le groupe et leur intérêt furent excellents.

Il sembla y avoir un accroissement de la moralité à l'hôpital et une amélioration de l'anxiété et de la dépression éprouvées par certains malades, et une diminution dans le nombre des malades atteints de crachats renfermant des bacilles de Koch. Des modifications apparurent dans les habitudes de l'hôpital et dans l'attitude du personnel.

Le chef de groupe devrait être soit un psychiatre, soit une personne spécialement entrainée professionnellement. Pour assurer le succès du traitement par groupe, il est nécessaire que l'administration de l'hôpital s'y intéresse, et ne soit pas relativement trop formelle. Il faut qu'elle ait le désir d'adopter une attitude scientifique.

Il est possible, grâce au traitement par groupe, d'atteindre un plus grand nombre de malades, avec une perte de temps et un effort relativement minimes. Le but de ce type de traitement de groupe n'est pas de guérir les

malades de leurs névroses, ni même de las rendre heureux, mais de les aider à s'imposer les conditions nécessaires de la vie en commun du sanatorium. Il s'agit d'une technique dont les limites et les avantages sont précis, et qui ne peut être appliquée à tout malade.

Ce sont les malades eux-mêmes beaucoup plus que le thérapeute qui s'aident les uns les autres dans les groupes. L'acceptation réelle d'être membre d'un petit groupe permet plus facilement d'accepter de faire partie du groupe étendu de l'hôpital. Il est possible aussi qu'elle permette une acceptation plus naturelle de la maladie et de sa signification pour les malades, leur famille et leurs amis.

Il s'agit d'un rapport préliminaire et l'auteur ne demande qu'une chose, c'est que des études complémentaires soient entreprises dans différents sanatoriums. Le programme tel qu'il a été conçu pourrait être mis en pratique avec d'autres malades, ne relevant pas de la psychiatrie obligés de séjourner à l'hôpital.

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Treatment of Tuberculosis in a Neuropsychiatric Hospital*

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The prevalence of tuberculosis in a Hospital for the mentally ill is an accepted fact. Anderson[1] in a recent review of this problem cited prevalence rates of 30 to 50 per thousand for psychiatric hospitals generally. Tompkins[2] reports prevalence rates in all Veterans Administration Neuropsychiatric Hospitals for World War I veterans of 9.5 per thousand and World War II veterans of 4.3 per thousand. The prevalence rate among all in-patients at our hospital has averaged 4 per thousand for the past five years and 3 per thousand for 1951. The latter figures cannot be compared with those from other hospitals for we have not included in this group patients having acquired pulmonary tuberculosis prior to admission to this hospital. However, these figures suggest that an active case-finding program in psychiatric hospitals in conjunction with a complete treatment program can be expected to result in decreasing prevalence rates in these hospitals, rates which can be expected to approach the rate for the general population.

Case-finding is the most important procedure in the program for control of tuberculosis, but it is of little value unless active cases brought to light are immediately isolated on a ward which fulfills all criteria for good aseptic techniques. All facilities must also be available for any indicated active therapy for both the tuberculosis and the particular mental illness. Further, these patients should not be permitted to return to a general psychiatric ward until the criteria for inactivity, as established by the National Tuberculosis Association, are fulfilled.

Difference of opinion exists as to whether or not these patients can be treated by the same criteria as are acceptable for treating non-psychotic patients. In the past, some few have believed that the treatment of these patients differed in no way from the treatment of the non-psychiatric patient with tuberculosis.[3,4] On the other hand, others felt that even pneumothorax was not suitable for mentally ill patients.[5] Radical chest surgery was not attempted by some,[6] whereas others[7] carried out all forms of collapse therapy but reserved major chest surgery for patients with "a favorable prognosis for remission of psychoses or those who have made a satisfactory adjustment with maximum privileges and minimal supervision." Recent reports[8-11] indicate a trend to more active therapy. This has been the result of finding that not only can psychiatric tuberculous patients be successfully treated in the same way that non-psychotic patients

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are treated, but that failure to utilize this type of therapeutic approach leaves the mental patient as a serious source of infection for other patients.

Five years ago, a tuberculosis neuropsychiatric unit was established in this hospital to care for an in-patient population of approximately 2,000 neuropsychiatric patients. It was planned to provide simultaneous active therapy for the patients' tuberculosis and mental illness. One hundred twenty seven cases of active tuberculosis have been treated during this period. Although final results are not available, the experience gained shows that many of the problems which were formerly considered as deterrents to active therapy can be resolved by the close supervision of the internist, psychiatrist, and thoracic surgeon.

Patients found on x-ray surveys to have lesions suspected of being tuberculosis should be isolated for study on the tuberculosis ward irrespective of mental reactions. They are usually found to be asymptomatic insofar as their tuberculosis is concerned. It is to be noted in this connection that mentally ill patients frequently do not complain of distress even though they are quite ill physically, whereas others may offer symptoms that may be mistaken for those of somatic illness but actually have no somatic basis.[12]

It is generally accepted that the physical examination is of little help in the mentally normal patient in evaluating the extent of tuberculosis. It is of less help in the uncooperative psychotic patient. One must also be aware of the fact that in many instances, the sputum examinations are not satisfactory since the obtaining of adequate specimens requires some cooperation on the part of the patient, and this is not always given by the psychotic patient. Therefore, the routine use of gastric washings becomes necessary. Also, bronchoscopic examinations should be performed in most cases to complete the studies. We have been able to do these without difficulty through the use of mild sedation and local anesthesia.

Specific bed-rest therapy cannot usually be obtained in psychotic tuberculous patients. Excessive physical activity in these patients can be curtailed by occupational therapy, group psychotherapy, and various types of sedentary entertainment in the form of movies, audience participation entertainment, ward parties and television. Religious services should be made available on the wards. A few hyperactive patients will require hydrotherapy, neutral pack treatment, or maintenance electroshock. An exceptional problem is presented in the catatonic, stuporous patients. These patients will remain at absolute bed rest, but this is of little real value in treating the tuberculosis because the catatonic state precludes an adequate intake of food. Electroconvulsive therapy, to relieve or alleviate the stupor, is indicated in this type of patient. In general, about 10 per cent of our patients can be encouraged to take bed rest as prescribed. About 40 per cent of them will rest for two hours in the morning and two hours in the afternoon. The remainder fall into the hyperactive or uncooperative group, and their activities may be curtailed by the adjunctive therapies mentioned above.

Pneumothorax and pneumoperitoneum can be given to most psychotic patients without difficulty. The uncooperative, hyperactive patient can be

treated with these measures after he has received electroconvulsive therapy and has become more easily manageable.

The indications for major surgery, thoracoplasties, or resections are the same as in a non-psychotic patient. The type of thoracoplasty operation used by us seemed especially adaptable for psychotic patients. Modified muscle-split thoracoplasty is routinely performed, using small incisions at different sites at each stage, thus obviating the necessity of re-opening any wound and insuring more adequate and rapid wound healing. The removal of seven ribs in three stages is routine. This type of thoracoplasty, because of the limited amount of surgical trauma in each stage, permits the subsequent stages to be performed at weekly intervals so that surgical intervention may be completed in three weeks.

It is not feasible to transfer psychotic patients to a general hospital or a tuberculosis hospital for surgery. The patient often becomes disturbed in strange surroundings and with strange personnel, particularly where

TABLE I: THERAPY USED IN TREATING PULMONARY TUBERCULOSIS IN 121 PSYCHIATRIC PATIENTS

I. *Rest Only in 52*		
II. *Special Procedures in 69*		
1. Pneumothorax (10 not therapeutically effective)	35	
2. Pneumonolysis	14	
3. Pneumoperitoneum (6 not effective)	29	
4. Phrenemphraxis	15	
5. Thoracoplasty (9 patients)	27	stages
6. Pneumonectomy	2	
7. Lobectomy	4	
8. Segmental Resections	2	
9. Decortication	3	

TABLE II: PULMONARY TUBERCULOSIS IN 121 PSYCHOTIC PATIENTS
(Preliminary Results 5 Year Period, 1947-1952)

Extent of Disease	Progressive	Improved	Arrested	Inactive	Died
Minimal, 14		1	1	12	
Moderately advanced, 78	2	35	12	22	7
Far advanced, 29	5	7		3	14
TOTAL, 121	7	43	13	37	21*

* 7 Patients were in a terminal stage of tuberculosis on admission.
7 Patients died of non-tuberculous conditions.
7 Patients, despite treatment, progressed and died.

TABLE III: ELECTROSHOCK IN 27 PSYCHOTIC PATIENTS WITH PULMONARY TUBERCULOSIS
(Preliminary Results 5 Year Period, 1947-1952)

Initial Status of Tuberculosis		Preliminary Results		
		Improved	Arrested	Inactive
Active	24	6	5 (1 relapse)	13 (1 relapse)
Arrested	3		2	1

there is no psychiatric orientation. We, therefore, strongly urge that necessary surgical facilities exist in the parent mental hospital.

We have, as noted above, treated active tuberculosis in 127 psychotic patients during the five years 1947 to 1952. We have not included patients who following appropriate studies, were found initially to have inactive lesions. One hundred twenty one cases had active pulmonary tuberculosis, and the remaining six included three cases of bone tuberculosis, one of glandular tuberculosis, one renal tuberculosis, and one tuberculous psoas abscess.

Table I shows the type of therapies used in treating 121 psychotic patients with pulmonary tuberculosis.

Table II lists the results in the pulmonary cases. It should be noted that these are preliminary reports as some of the cases have been under treatment a relatively short time. Nevertheless, the table gives indication as to what can be expected in the treatment of the tuberculous neuropsychiatric patient.

The use of electroshock therapy in treating psychiatric patients with associated pulmonary tuberculosis should no longer be considered a problem in psychiatric hospitals. One of us reviewed this problem a few years ago, reporting case material in detail,[13] and concluded that "cases reported in the literature as developing tuberculosis following electroshock therapy had not been adequately controlled by x-ray film or clinical studies to an extent warranting definite conclusions." Earlier observations by Moore,[14] Kalinowsky and Hoch[15] indicated that patients with active tuberculosis could receive electroshock without danger of spreading their tuberculosis. Jeftoft[16] and, recently, Clark[17] have shown electroconvulsive therapy can be given without adverse effect on healed or active lesions. Clark[17] reported on 110 such cases and found no evidence of any adverse effect. He recommended that "further long term studies of treated cases are most desirable." Since our initial report, we have treated additional cases with the result shown in Table III. Altogether 27 patients have received electroconvulsive therapy at some time during the period of treatment for their tuberculosis. The table indicates the stage of their tuberculosis at the time the shock was given and their present condition. These 27 patients have received a total of 1,273 electroshock treatments. Details of the two cases showing relapses are given below:

Case 1: C.S. This 25 year old white male veteran of World War II was admitted to the hospital January 5, 1945 with a diagnosis of schizophrenia, mixed type. On September 1, 1948 a routine chest x-ray film showed a minimal tuberculous lesion of the left upper lobe. Sputum examinations were negative but gastric washings were positive for acid-fast bacilli. He was treated by bed rest, but it was necessary to give him electroshock therapy in order to gain his cooperation. The tuberculosis improved and by January 1950 had met the criteria for inactivity. From September 1948 to June 1950 he received a total of 91 electroshock treatments. On April 18, 1951, chest x-ray film showed an increase in the size of the lesion at the left upper lobe. Planigraphic studies revealed a small cavity. A gastric washing was positive for acid-fast bacilli. This relapse occurred nine months after the last electroshock treatment. Subsequently, he received streptomycin and para-aminosalicylic acid, and finally segmental resection was performed. The resected specimen consisted

of a small fibrotic mass with a central thick-walled cavity 1 cm. in diameter. His postoperative course has been good, but it is too early to give the final results.

Case 2: N.B. This 24 year old white male veteran was admitted to the hospital May 15, 1934 with diagnosis of schizophrenia, simple type. A routine chest x-ray film March 1947 showed evidence of a minimal lesion in the left upper lobe. Sputum and gastric washings were negative for acid-fast bacilli. There was no symptom. Sedimentation rate was normal. He was uncooperative in taking bed rest; however, in spite of this, the lesion became smaller and appeared fibrotic. By June 1950 the disease was classified as minimal, arrested. In October 1950 he was started on electroconvulsive therapy because his mental condition seemed worse. In June 1951 the chest x-ray film showed some increase in the left upper lobe lesion. Sputum studies were negative for acid-fast bacilli, but a gastric washing was positive. Electroshock therapy was continued until August 1951, and was discontinued when his mental condition showed some improvement. He received a total of 39 treatments. He has now been given pneumothorax on the left side, and with the aid of a pneumonolysis has a good collapse. Sputum studies and one gastric washing have been negative on culture and guinea pig innoculation.

We do not believe electroshock should be considered a factor in the relapses of these two patients. We are of the opinion that the initial lesions, although appearing stable on x-ray film inspection had remained active, and the subsequent changes on x-ray films represent the normal progression of the tuberculosis. Their current treatment is more definitive, and we anticipate better results.

Many of these patients, in addition to the initial course of electroconvulsive therapy three times a week for six weeks or a total of 18 treatments, required weekly maintenance electroshock therapy. This was necessary in order to gain the patients' cooperation in taking treatment and adequate nourishment. One patient with moderately active disease received a total of 142 electroshock treatments in order to gain his cooperation in taking pneumothorax and sufficient nourishment. His tuberculosis has been inactive for one year.

The decision to give electroconvulsive therapy to psychotic patients with tuberculosis is based upon weighing the anticipated advantages against the risks. For example, in a tuberculous psychotic patient, who is acutely disturbed, physically hyperactive, and refuses to eat, the chances are that these symptoms will result in a spread of the tuberculous lesion. Shock can be expected to make this patient less physically active, more amenable to suggestion, and to improve his appetite. In all cases, the decision to use shock is reached by professional collaboration and agreement between the internist and the psychiatrist. The indications for giving electroshock in the presence of tuberculosis can be summarized as follows:[13] "(1) Acute psychiatric illnesses with associated tuberculosis, if delaying the shock therapy would reduce the possibilities for a good prognosis in the mental illness. (2) Whenever there is a possibility of alleviating symptoms which interfere with treatment of the tuberculosis. This refers principally to gaining cooperation of the patient in obtaining proper nourishment, bed rest, or collapse therapy where indicated."

No discussion of the tuberculosis-neuropsychiatric problem is complete without mention of the part played by the nursing service. We believe that

proper control and care for these patients would be impossible without nurses trained in psychiatric, medical, tuberculosis, and surgical nursing. The nurse in charge of the tuberculosis psychiatric ward should be qualified in all of these nursing specialities. She should be sufficiently able to take care of patients' needs so that it is not necessary to bring in strange nurses or transfer patients to other sections of the hospital for special procedures. Once a psychiatric patient becomes accustomed to the same ward nurses and personnel, cooperation is definitely enhanced. Neuropsychiatric tuberculosis nursing should be recognized as a highly specialized branch of nursing, requiring combined skill in specialized nursing.

SUMMARY

1) Our experience suggests that the high incidence of tuberculosis in hospitals for mental patients can be reduced by a continuous case finding program followed by adequate therapy of active cases.

2) Neuropsychiatric patients with tuberculosis should receive all types of therapy available to mentally normal patients inclusive of major surgery. The indications for temporary and permanent collapse procedures and resections are the same as in the non-psychotic patient.

3) The patients' mental conditions should be treated concurrently and electroconvulsive therapy should be used when indicated in the presence of active tuberculosis.

4) It is emphasized that the treatment of neuropsychiatric tuberculous patients requires the active supervision of the internist, psychiatrist, thoracic surgeon and nurses trained and experienced in caring for them.

5) Preliminary data in the treatment of 121 psychotic patients with active pulmonary tuberculosis confirm these conclusions.

RESUMEN

1) Nuestra experiencia sugiere que la elevada frecuencia de la tuberculosis en los hospitales para enfermos mentales puede reducirse por medio de un sistema contínuo de investigación seguido de una terapéutica adecuada de los casos activos descubiertos.

2) Los enfermos mentales con tuberculosis deben recibir todas las formas de tratamiento que hay para los enfermos mentalmente normales incluyendo la cirugía mayor. Las indicaciones para los métodos de colapso temporal y permanente así como las resecciones son las mismas que para los enfermos no psicóticos.

3) Los padecimientos mentales deben tratarse al mismo tiempo y la terapéutica convulsivante eléctrica debe usarse cuando esté indicada, en presencia de tuberculosis activa.

4) Se reclaca que el tratamiento de los enfermos neuro-psiquiátricos tuberculosos requiere la supervisión activa del internista, el psiquiatra, el cirujano de torax y de las enfermeras preparadas para estos cuidados de ellos.

5) Los datos preliminares de 121 enfermos neuro-psiquiátricos con tuberculosis activa confirman estas conclusiones.

RESUME

1) Les auteurs, d'après leur expérience, pensent que le grand pourcentage de tuberculose dans les hôpitaux psychiatriques peut être réduit par une recherche systématique ininterrompue de ces cas suivie du traitement convenable des formes actives.

2) Les malades de neuropsychiatrie atteints de tuberculose devraient être traités exactement dans les mêmes conditions que les malades mentalement normaux, y compris par la grande chirurgie. Les indications de la collapsothérapie temporaire ou permanente et des exérèses sont les mêmes que chez les malades n'ayant pas de troubles psychiques.

3) L'affection mentale des malades devrait être traitée parallèlement et on peut utiliser l'électro-choc quand il est indiqué, malgré l'existence d'une tuberculose active.

4) Les auteurs insistent sur le fait que le traitement de la tuberculose chez les malades atteints d'affections neuropsychiatriques demande un contrôle actif du médecin de médecine générale, du psychiatre, du chirurgien thoracique, et d'infirmièrimentées dans les particularités qui concernent leur traitement.

5) Les résultats préliminaires obtenus par le traitement de 121 tuberculeux atteints de troubles psychiques sont la confirmation de ces conclusions.

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The Relationship Between Pathological Changes in Blood Vessels in Resected Lobes and Lungs as Correlated with Pulmonary Artery Pressure Changes Recorded During Cardiac Catheterization*

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Because of the dearth of accurate information of the pathological variations in the pulmonary vessels in tuberculosis of the lungs, a study was undertaken in 85 cases where lobectomy and pneumonectomy had been performed. In the course of this study[1] an attempt was made to correlate these pathological changes with data derived from a small group of the series in the cardio-pulmonary laboratory, particularly with regard to the pulmonary artery pressures at rest and exercise. Although data on the cardiac output and arterial saturation in these cases are included, no attempt has been made to evaluate any variations in these findings from the norm in this paper.

Sections were taken not only from the tuberculous portions of the specimen, but from uninvolved portions as well. Routine stains as well as elastic fibre stains were used. While a quantitative approach to the degree of involvement of the vessels could not be made, changes were described as slight, moderate, and severe, and a particular attempt was made to classify separately the variations in the different sized arteries and arterioles.

In summary, the large arteries showed varying degrees of fibroblastic proliferation of the intima usually diffusely or concentrically located and sometimes obstructing the lumen. The medium-sized vessels showed similar changes. The small arteries, in addition to a chronic endarteritic process, often showed scarring or edema of the media and infiltration of the entire wall by lymphocytes and some polymorphs. The arterioles especially in peribronchial locations exhibited uniform fibrocytic, severely obstructive, intimal proliferation. These changes were observed not only in the walls of cavities but were widespread in areas of parenchymal fibrosis, bronchial fibrosis, and areas of chronic, apparently non-specific interstitial inflammation. In relatively normal portions of the lung, remote from the active tuberculous processes, the very small arterioles and precapillaries frequently showed intimal hyalinization or fibrosis of the wall.

Of the 85 cases studied pathologically, only 17 were available on whom adequate data had been secured during cardiac catheterization. There were 11 females and six males ranging in age from 20 to 45. The table

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Name	Number	Spec.	VASCULAR CHANGES ARTERIES	ARTERIOLES
L.O.	49S-34	RUL	Minimal	Occasional slight thickening of walls in partially collapsed areas.
J.K.*	49S-29	RUL	Localized diffuse changes related to active nodules.	Minimal.
M.W.	49S-13	LL	Marked extensive obstructive changes large and medium vessels both in cavity wall and extensive fibrosis.	Extensive moderate thickened walls in cavity wall and peribronchial fibrous tissue elsewhere.
L.V.	50S-23	RL	Severe changes small vessels in fibrous and collapsed lung.	Extensive severe changes in collapsed lung and interstitial fibrosis.
A.F.*	49S-32	LL	Moderate thickening occasionally obstructive in medium vessels related to bronchitis.	Minimal.
S.S.*	50S-6	RL	Extensive obstructive changes in smaller vessels in relation to caseation.	Moderate diffuse sclerosis in remants of collapse parenchyma.
R.M.*	50S-11	RML LL	None	Extensive moderate changes between miliary foci.
H.F.	50S-43	RL	Occasional slight changes in wall of cavity.	Moderate extensive changes in normal collapsed lung.
D.L.*	50S-13	LUL	Moderate to severe extensive changes in all areas of collapse, caseation and fibrosis.	Moderate changes in relation to areas of collapse, fibrosis and caseation.
F.G.	50S-48	LUL	Moderately severe and extensive in the cavitary wall and in the non-specific fibrotic, and inflammatory changes.	Moderate in severity and extent.
R.L.	50S-14	LLL	Moderate changes in inflammed and nearby areas.	Moderate in extent and severity plus changes in relatively normal tissue.
A.D.	50S-29	LL	Moderate in severity and extent in large vessels, slight in small vessels.	Moderate in severity and extent. Veins show changes in normal lung tissue.
B.P.	50S-39	LL	Severe and extensive changes near nodules, as well as fibrous and inflammed areas, veins involved.	Extensive and severe throughout.
L.W.	50S-44	RL	Extensive moderate changes in nodule and fibrotic areas.	Extensive moderate to severe in collapsed areas.
M.V.	51S-3	LL	Small and slight changes moderate in extent in areas of tuberculosis.	Moderate changes slight extent.
D.L.	50S-53	LLL	Severe changes in small arteries in cavity wall and in the fibrotic interstitial tissues.	Normal vessels in normal tissue.
K.W.	50S-37	RUL	Moderate, not extensive in both caseous areas as well as interstitial pneumonitis.	Moderate, not extensive.

TABLE (Continued)

Name	Number	Spec.		RESTING Pul. Art. Pressure Insp.	Exp.	EXERCISE Pul. Art. Pressure Insp.	Exp	CARDIAC OUTPUT Rest	Exer.	ARTERIAL SATURATION Rest	Exer.	MAXIMUM BREATHING CAPACITY
L.O.	49S-34	RUL		34/20	42/20	70/40	83/42	4.8L	7.4L	84%	85%	89L/min.
J.K.*	49S-29	RUL		42/2	48/5			7.9L		91%		83L/min.
M.W.	49S-13	LL		33/1	45/7			4.6L		92%		44L/min.
L.V.	50S-23	RL		20/6	28/13	29/13	43/21	4.4L	6.4L	93%	94%	42L/min.
A.F.*	49S-32	LL		24/0	28/2			5.6L	7.8L	97%	94%	108L/min.
S.S.*	50S-6	RL		18/8	27/0			4.8L		83%		34L/min.
R.M.*	50S-11	RML LL		19/8	23/7	27/10		5.5L	6.8L	89%	97%	65L/min.
H.F.	50S-43	RL		22/2	25/3			6.4L		92%		132L/min.
D.L.*	50S-13	LUL		26/0	34/4	51/3	68/15	6.4L	13.7L	91%	88%	44L/min.
F.G.	50S-48	LUL		20/5	34/12	34/9	45/25	4.8L	6.4L	91%	93%	47L/min.
R.L.	50S-14	LLL	Pre	13/6	18/8	18/8	28/10	4.6L	6.1L	90%	95%	51L/min.
			Post	20/0	26/6	32/6	44/10	3.9L	6.4L	90%	96%	60L/min.
A.D.	50S-29	LL	Pre	18/8	28/13	21/8	20/13	4.1L	5.5L	93%	97%	67L/min.
			Post	19/8	25/10	31/10	44/20	3.6L	5.9L	91%	94%	60L/min.
B.P.	50S-39	LL	Pre	21/4	27/11	34/6	57/27	4.3L	7.0L	91%	92%	44L/min.
			Post	14/4	21/8	24/12	36/20	3.2L	6.15L	91%	91%	49L/min.
L.W.	50S-44	RL	Pre	20/5	26/9	38/11	48/21	4.0L	7.3L	95%	89%	58L/min.
			Post	25/6	27/11			4.6L		88%		53L/min.
M.V.	51S-3	LL		21/4	22/8	19/8	24/12	4.1L		93%		56L/min.
D.L.	50S-53	LLL	Pre	14/4	16/5	22/3	32/12	5.6L	11.8L	96%	93%	101L/min.
			Post	17/6	18/8	17/9	23/16	5.2L	7.6L	88%	91%	85L/min.
K.W.	50S-37	RUL		22/9	25/12	29/8	37/11	6.6L	9.0L	94%	96%	138L/min.

*Right ventricular pressures taken instead of pulmonary artery.

shows the history, the vascular changes in the arteries and arterioles are correlated with the pulmonary artery pressures on rest and exercise, the cardiac output and arterial saturation on rest and exercise, and the maximum breathing capacity. Bronchospirometric figures were not included. In five cases, postoperative studies were available and have been included. In six cases, the recorded pressures are from the right ventricle rather than the pulmonary artery.

It should be noted from the table that there is little correlation between the pathologic changes in the arteries and arterioles and the catheterization findings. As an example, case L.O. (1) showed minimal changes in the arteries with only slight thickening of arteriolar walls in partially collapsed areas. Her pulmonary arterial pressures were high at rest and increased more than twice on exercise. This variation from the anticipated is based on the extensive contralateral disease present. Similarly in case J.K. (2) the high resting pressures are not associated with slight changes in the vascular structures for the same reason. Case L.V. (4) shows a closer approximation to the true picture of correlation of severe changes in the vessels and normal pulmonary arterial pressures at rest rising on exercise. There was no manifest contralateral disease and the lung to be resected had been collapsed by thoracoplasty for several years. Any attempt at correlation therefore was doomed to failure by virtue of the fact that tuberculosis rarely is a localized disease and any changes present in the contralateral lung bring equivalent vascular changes.

Discussion

The pulmonary vascular bed is characterized by its extreme distensibility. Hamilton[2] and others have pointed out that about three times the resting volume of blood in the bed can be accommodated without an appreciable rise in pulmonary arterial pressure. Pressure in a fluid system is the result of volume flow times resistance, and since this is the case the resistance in the pulmonary circuit under normal circumstances is low. This is borne out by the findings of Hickman and Cargill[3] which showed that in a series of normal males pulmonary arterial pressures did not rise with moderate exercise though the cardiac output was increased.

The factors involved in the regulation of pulmonary arterial pressure are still complex and difficult to evaluate. Anatomically (Drinker[4]) nerve fibrils can be shown to reach the pulmonary capillaries but vasoconstrictive and vasodilatative effects are not marked when these are stimulated pharmacologically as with histamine.

In cats, von Euler and Liljestrand[5] have shown that the breathing of 100 per cent oxygen caused a marked drop in the pulmonary arterial pressure, the cardiac output being unaltered as judged by the pulse rate. Recently Motley and others[6] showed that the breathing of anoxic mixtures such as 10 and 12 per cent oxygen-nitrogen caused pulmonary hypertension. Dressler et al.[7] showed that pulmonary arterial pressures dropped on breathing 100 per cent oxygen, with the cardiac output remaining unchanged. The quoted work has been taken to mean that oxygen should

have a direct effect on the pulmonary vascular bed causing vasodilation in well oxygenated areas while the converse should be true in poorly ventilated areas. Actually, this point is still highly debatable.

Cournand[8] has pointed out that under normal conditions the pulmonary circulation is regulated mechanically by the fluctuations of intrathoracic pressure during the various phases of respiration. In normal quiet respiration no marked effect is noted on the pulmonary pressure. In deep breathing a marked effect is noted in that in inspiration with highly negative intrathoracic pressure the inflow of blood into the right heart is increased with a resultant increased right ventricular discharge or increase in mean pulmonary arterial pressure, according to Starling's Law. The left ventricular discharge is decreased during this period since less blood is being displaced from the lungs into the left heart. In expiration the reverse is true.

If then the pulmonary circulation is under control of the various mechanisms listed above, the causes of pulmonary hypertension can be ascribed to some of the following reasons:

1) Increase in peripheral resistance due to obliteration of the vascular bed or loss of elasticity contributes certainly to the development of pulmonary hypertension. Increase in vascular tone of the pulmonary vessels may increase peripheral resistance and so may cause pulmonary hypertension. An increased blood volume causes hypertension only if associated with the above phenomena.

2) Anoxia causing vasoconstriction as described by von Euler and Liljestrand.

3) Inadequate emptying or filling of the left heart such as in mitral stenosis, left ventricular failure, etc., which causes an increase in pulmonary capillary pressure and so pulmonary arterial hypertension.

Of the 17 cases studied, four showed hypertension at rest. Of 12 cases which were exercised pre-operatively, six developed hypertension. Post-operatively five cases were studied. None of these showed hypertension at rest, while four developed it after exercise.

These findings are easily explained on the basis of a reduced vascular bed and increased peripheral resistance. It would appear that for the most part, the average tuberculous patient facing resection in this hospital still has a vascular bed sufficient to produce normal pressures at rest. On moderate exercise, a large proportion of these patients cannot accommodate the increased blood flow and cardiac output without a simultaneous rise in pressure. The role of local tissue anoxia cannot be satisfactorily evaluated. Cournand has pointed out the lack of correlation which occurs in chronic pulmonary disease as regards ventilation-perfusion relationships. The question of whether local anoxia plays a significant role in terms of local blood flow is still debatable. In addition, the pathological changes in the vessels in these areas may be sufficient to limit the degree of possible vasoconstriction and vasodilatation.

In those five cases studied pre- and post-operatively, the findings are not as easily explained. Cournand et al.[9] has stated that patients who have had pneumonectomy must of necessity accommodate the same quantity

of blood formerly handled by both lungs in the one remaining lung. Such individuals react to mild exercise with pulmonary hypertension of the magnitude ordinarily found in normal individuals under severe exercise. Mendelsohn et al.[16] studied the pulmonary hemodynamics during pulmonary resection. They found that while the pulmonary arterial pressures rose after ligation of one major vessel, they soon returned to normal. The one exception was in a case of severe emphysema where a rise in tension developed and persisted until death after a short time with cor pulmonale.

It would be expected therefore that in tuberculous individuals where the vascular bed is essentially normal on the uninvolved side, that the pressures post-operatively would be normal at rest, and rise on moderate exercise. It is true that four of the five cases showed a rise after exercise. It is to be noted, however, that two of the cases started their exercise pressure rise from a lower level in the post-operative state. This would indicate some unexplained mechanism which caused an over-all drop in pressure which should otherwise have increased if the previously postulated explanation held true.

SUMMARY

During the past few years we have witnessed an ever growing knowledge of the cardio-pulmonary system. It has become possible to explain on a sound physiological basis many of the hitherto obscure phenomena concerned in the regulation of the pulmonary circulation. The exact significance of much of the data gathered in studying chronic pulmonary disease, however, still cannot be correlated satisfactorily. While it may be concluded that the disease alterations of the vascular bed must play a large role in the development of pulmonary hypertension, its exact position in the problem cannot be assessed adequately.

RESUMEN

Hemos presenciado en los últimos años un conocimiento siempre creciente del aparato cardiopulmonar. Asi ha sido posible explicar algunos fenómenos que hasta aqui eran obscuros, y la explicación se puede establecer sobre bases fisiológicas sólidas, tratándose de la regulación de la circulación pulmonar. La significación exacta de muchos de los datos acumulados al estudiar la enfermedad crónica pulmonar sin embargo, no puede interpretarse satisfactoriamente. Si bien se puede concluir que las alteraciones patológicas del lecho vascular deben desempañar un papel importante en el desarrollo de la hipertensión pulmonar, su posición exacta dentro del problema no puede estimarse de modo adecuado.

RESUME

Dans ces dernières années, on a assisté à un accroissement constant de nos connaissances concernant le couple cardio-pulmonaire. L'explication physiologique de beaucoup de phénomènes concernant la circulation pulmonaire qui jusqu'ici semblaient obscurs, est maintenant devenue possible. Toutefois, la signification exacte de nombreux faits récoltés lors de l'étude

des affections pulmonaires chroniques reste encore incertaine. Sans doute, on peut conclure que les altérations pathologiques du lit vasculaire jouent un rôle important dans le développement de l'hypertension pulmonaire, mais le problème n'est pas encore résolu d'une façon précise.

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Discussion

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Respiratory function depends on an adequate correlation of ventilation and perfusion of the lungs. Studies of the pulmonary circulation are of great importance in improving our understanding of the alterations associated with pulmonary diseases. Various approaches to the problem are required as no one single method gives data which is conclusive. Cardiac catheterization has been a most important addition to the technics of physiologic study. It has a great advantage over injection studies of the pulmonary vessels because blood flow rather than the mere size of the vascular channels is the important factor from the standpoint of function.

In pumonary tuberculosis the reduction in respiratory function is more contingent on a diffuse distribution of the pathologic process rather than the presence of advanced cavitary disease in a localized area. An old bilateral disseminated lesion which has largely cleared radiographically may have reduced respiratory function considerably more than the total destruction of one or two lobes. Therefore the crucial point in pulmonary function after collapse therapy or resection is the ventilatory and circulatory status in the remaining pulmonary tissue. As the authors have indicated, the pulmonary artery pressure depends largely on the size and

distensibility of the total pulmonary vascular bed rather than the extent of the obliteration of the pulmonary vessels in the area of major disease. It is therefore not unexpected that there was no close correlation between the vascular changes in the resected lobes and the alterations in pulmonary artery pressure. It should also be noted that in several instances in which the post-operative pulmonary artery pressure on exercise was lower than the pre-operative figure, the cardiac output was also lower. Therefore the reduced minute blood flow through the lungs may have been an important factor in the lower pulmonary artery pressure. The authors have wisely refrained from attempting to draw many conclusions at this time. Their interesting and provocative work points to one of the ways in which our further basic knowledge of pulmonary disease will be acquired.

Finally it should be borne in mind that in some pulmonary and cardiac diseases there are communications of considerable magnitude between the pulmonary and bronchial arterial systems which further complicate physiologic measurements and are of clinical significance.

Arterio-Venous Aneurysma of the Lung

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Most of the characteristic features of arterio-venous aneurysm of the lung are presented by a case observed in the Opland County Hospital.

Case Report

O.S., a 49 year old man was admitted to the surgical department with osteomyelitis in his left tibia on November 5, 1951.

When 20 years old, he had pleurisy on the right side. X-ray films during the following years revealed nothing abnormal. In 1936 he was treated for acute appendicitis. No remark was made then about cyanosis or clubbing of the fingers. The postoperative course was uneventful.

The osteomyelitis was treated with incision and penicillin and he made a rapid recovery.

During hospitalization he was referred to the medical department because of pronounced cyanosis and clubbing of the fingers.

The family history was negative. Particularly there was no evidence of Rendu-Osler-Weber's disease. On questioning he admitted slight shortness of breath since childhood, but could easily manage to go hunting in the mountains at a moderate pace. He believed his fingers had always been clubbed. One week before admission he had a fit with loss of consciousness and convulsions. There was complete amnesia. He had never had hemoptysis.

On examination he was a slender man with marked cyanosis and clubbing of the fingers and toes. There were no hemangiomas in the skin or the visible mucous membranes. Over the base of the left lung near the spine a slight systolic murmur was heard during inspiration. The heart was normal on clinical examination. Blood pressure was 120/80. With the diagnosis of arterio-venous aneurysm in the lower left lobe he was referred for radiographic examination of the chest. Postero-anterior and left lateral chest films showed a round shadow, the size of a golf ball, behind the heart. From the shadow, best seen in the planigrams, two enlarged vessels led to the pulmonary hilum (Figures 1, 2, 3 and 4).

The urine was normal. The hemoglobin was 22.2 Gm. per 100 ml. blood, red cell count 6,66 mill. per cmm., W.B.C. 6,700 with normal distribution in the Arneth count.

Surgical treatment was advised, but he wanted "to think it over," and was discharged on November 17.

Two days later he was readmitted as an emergency case with right hemiplegia. This was interpreted as cerebral thrombosis or cerebral embolism from the aneurysm. He was treated with heparin-dicoumarin, but went a rapid downhill course and died on November 28, 1951.

The necropsy revealed dense adhesions between the right lung and the thoracic wall. The right lung was otherwise normal. There were some thin adhesions between the left lung and the thoracic wall. Projecting from the diaphragmatic surface of the left lung was seen the aneurysm, the size of a thumb. The wall was paper-thin and had a blue-black color with some white spots, probably fibrin. The artery and the veins of the left lung were injected with polyvenylacetic ester after washing with saline. The lung was then plunged into concentrated hydrochloric acid which digested all organic tissue leaving the vascular tree and the aneurysm intact (Figures 5 and 6).

The other organs were normal, except the thyroid gland which was enlarged

(weight 60 Gm.). The brain was fixed in formalin for a later examination which showed an abscess in the left hemisphere (3 x 4 x 3 cm.). No microorganisms were found on microscopical examination.

Considering the clear-cut clinical picture presented by arterio-venous aneuryms, it is peculiar that the diagnosis was made for the first time in 1939 (Smith and Horton). Since then an increasing number of cases have been reported. In 1949 Yater et al. collected 43 cases from the literature, and added two of their own. By reviewing the list it seems probable that

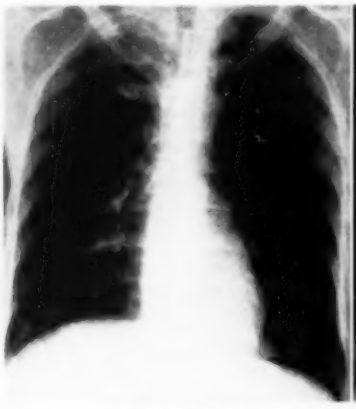

FIGURE 1

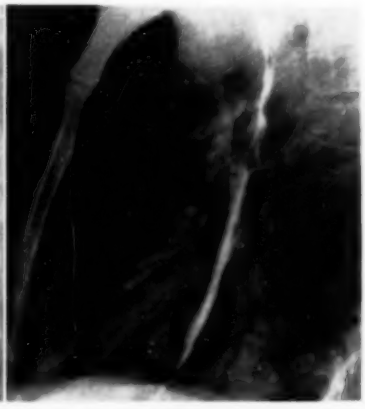

FIGURE 2

Figure 1: A-P projection of the chest showing the aneurysma in the left cardio-diaphragmatic angle.—*Figure 2:* The aneurysma is seen as a round shadow behind the heart.

FIGURE 3

FIGURE 4

Figure 3: Lateral planigram showing the aneurysma connected with the pulmonary hilum with two band-shaped shadows.—*Figure 4:* Oblique planigram showing the loop formed by the aneurysma and the dilated vessels.

one of these was tumor of the hemangioma type (37). Two other cases were in two different reports (24, 40). On the other hand some cases were overlooked. A minimum of 79 cases have now been reported in the literature, and certainly a number of unpublished cases have been diagnosed. A review of the reported cases and their main clinical characteristics are given in Table I.

The male-female ratio is 2 to 1. In one-half of the cases cutaneous teleangiectasies could be demonstrated, and in many teleangiectasies were present in the near relatives of the patients. Goldman supposes that arteriovenous aneurysm is a manifestation of hereditary hemorrhagic teleangiectasia (Rendu-Osler-Weber's disease, Goldsteins heredofamilial angioma-

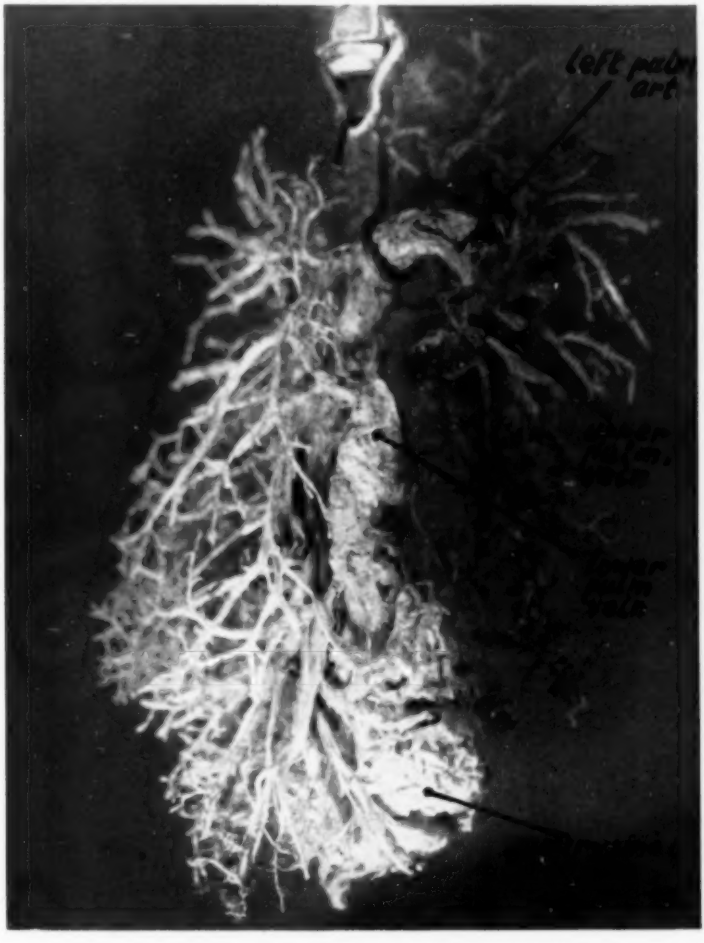

FIGURE 5: The lung-preparation (see text), medial view.

tosis). The clinical pictures in the two diseases are very different. Cyanosis and secondary polycythemia are characteristics of the former while anemia caused by recurrent hemorrhages is the dominating feature in the latter. However, overlapping may occur. Typical cases of Rendu-Osler-Weber's disease may have arterio-venous aneurysm, the symptoms of which are sometimes overshadowed by the anemia. In many such cases it seems probable that the aneurysm has been overlooked (14, 29 case 2, 30, 51).

The chief complaint in nearly all the patients was shortness of breath. Nervous symptoms in the form of dizziness, faintness or the feeling of numbness or weakness in one side of the body was a common occurrence. The cause of the nervous symptoms is not quite clear. Lindgren thought they might be due to small air emboli to the brain, but more probably they

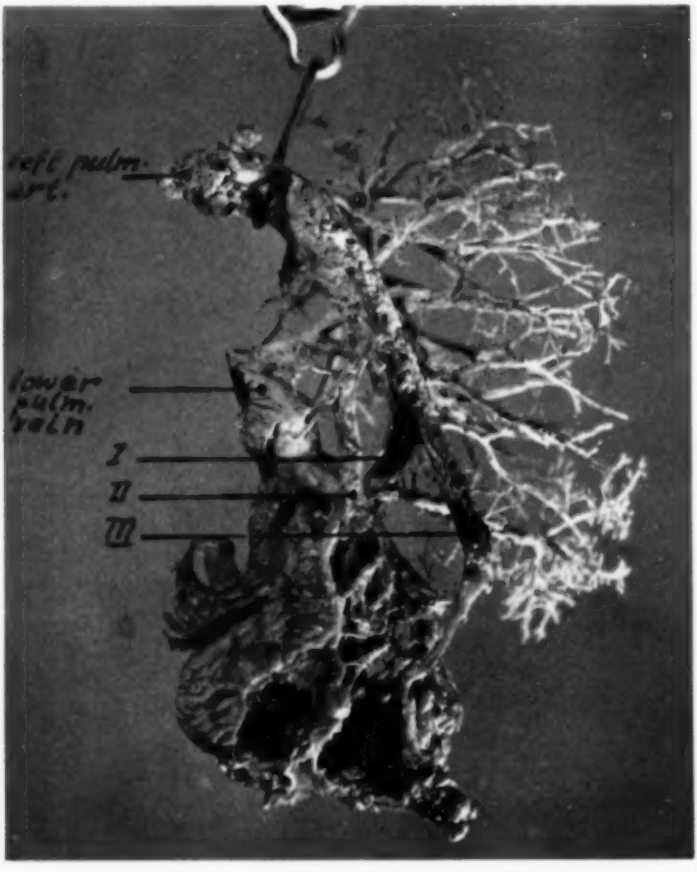

FIGURE 6: The lung-preparation, oblique view (the same projection as the planigram, Figure 4). The vascular branches not connected with the aneurysma are cut away. Three arterial branches enter the aneurysmal sack which is drained by a greatly enlarged vein.

are caused by local anoxemia in the central nervous system. Cyanosis is the most prominent objective sign. It may reach extreme degrees and is caused by the systemic arterial oxygen unsaturation. When a mean of about 5 Gm. hemoglobin per 100 ml. blood is present in reduced form in the capillaries, cyanosis ensues. In one of the cases reported by Friedlich et al., only 68 per cent of the arterial blood was saturated with oxygen. With a hemoglobin content of 25.3 Gm. this means about 8 Gm. reduced hemoglobin in the arterial end of the capillary loop. Eighty per cent of the blood flow in the lesser circulation may be shunted through the aneurysmal sack and so avoid oxygenation in the alveolar capillaries.[4] The arterial unsaturation will give rise to a secondary polycythemia with erythrocyte counts as high as 11.5 mill. per cm.[27] The polycythemia will cause a considerable rise in the circulating blood volume due solely to the augmentation of the cell mass, while the plasma volume remains normal. Thence the hematocrit readings will be high (90 per cent in the case of Baker and Trounce). In contrast to aneurysms in the systemic circulation, pulmonary arterio-venous aneurysms will not or very rarely cause cardiac hypertrophy. In the former an arteriovenous shunt will cause a considerable decrease in the vascular resistance and an increase in the minute volume. In the lesser circulation, the vascular resistance is normally so low that the presence of a shunt will not significantly reduce the overall vascular resistance of the lung. Friedlich et al., have found that the capillary resistance in the lung is higher than normal in the presence of pulmonary arterio-venous aneuryms. They mention two possible factors leading to increased resistance. The first may be multiple small thrombi occluding the lung capillaries as described in the tetralogy of Fallot. The second may be vascular constriction caused by anoxemia.

In 15 cases no mention is made of cyanosis or clubbing of the fingers. In some of these cases (11, 17, 30, 55) only brief notes are given of the clinical symptoms. In another (51) cyanosis might have been hidden by anemia caused by recurrent hemorrhages. In one case (59) the "feeder artery" came from the descending part of the aorta and there was no right-to-left shunt. In other cases without cyanosis the explanation may be that the shunt was from a bronchial artery to a pulmonary vein. In more than one-half of the cases a murmur could be heard over the aneurysm and sometimes (47, 61) a thrill could be felt. The bruit is most often systolic and is best heard during inspiration.

X-ray examination is of utmost importance for the diagnosis. In spite of the characteristic clinical picture, in nearly all the cases the first step towards a correct diagnosis was the finding of a shadow in the lung fields. Lindgren gives an excellent description of the x-ray findings: "The most important criterion of the nature of the change is its relation to the surrounding vessels. Even in the case of small aneurysms one sees at least two vessels in the vicinity which are broader than others situated in the same region and which are often especially conspicious in that they have another course than other vessels and are more sinuous." By the Valsalva test (inspiration against closed glottis) the shadow will augment, and

TABLE I

Case	Author	Sex	Age	Age at onset of symptoms	Family history	Heman-giomas	Cyanosis and clubbing	Bruit	Nervous symptoms	Hemop-tysis	Number of aneurysms	Course
1.*	Curton	M	12	11	—	—	—	+	—	+	Mult.	Death by hemoptysis
2.*	Hedenius	M	29	19	—	—	—	—	—	+	Sing.	Death after hemoptysis
3.	Wilkens	F	23	17	—	+	+	+	—	—	Mult.	Death by hemothorax
4.	Reading	F	4	-	—	—	+	+	—	—	Mult.	Death by brain abscess
5.	Bowers	M	2 days	-	-	—	—	—	—	—	Mult.	Death by hemothorax
6.	Rodes	M	25	1	—	+	+	—	+	+	Mult.	Death by hemoptysis
7.	Smith and Horton	M	40	1	—	+	+	+	+	—	Sing.	Unknown
8.	Hepburn and Dauphinee	F	23	15	—	—	+	—	+	—	Mult.	Cured by pneumonectomy
9.	Goldman	M	22	6	+	+	+	—	—	—	Sing.	Cured by pneumonectomy
10.	Adams et al.	M	24	8	+	+	+	—	—	—	Mult.	Cured by pneumonectomy
11.	Janes	M	30	29	—	+	+	+	—	+	Mult.	Cured by excision
12.	Jones et al.	F	24	1	—	—	+	+	—	—	Mult.	Cured by pneumonectomy
13.	Alexander et al.	M	41	-	—	—	+	—	+	—	Mult.	Death by coronary disease
14.*	Rundles	M	56	42	—	+	—	—	—	+	Sing.	Death by hemoptysis
15.	Sisson et al.	F	45	23	—	+	+	+	+	+	Mult.	Death by angiography
16.	Makler et al. and Friedlich et al.	M	21	14	—	+	+	+	+	—	Mult.	Cured by lobectomy and excision
17.	Lindgren	M	30	14	—	—	+	+	—	—	Sing.	Unchanged after 4 years

* Uncertain cases.

TABLE I (Continued)

Case	Author	Sex	Age	Age at onset of symptoms	Family history	Heman-giomas	Cyanosis and clubbing	Bruit	Nervous symptoms	Hemop-tysis	Number of aneurysms	Course
18.	Giampalmo	M	29	25	+	+	+	+	+	+	Mult.	Cured by lobectomy and excision
19.	Giampalmo	F	25	15	–	–	+	+	–	+	Sing.	Cured by excision
20.	Beierwaltes et al.	F	27	1	+	+	+	–	+	–	Sing.	Improved after lobectomy
21.	Bisgard	M	29	21	–	+	+	+	+	–	Mult.	Cured by lobectomy
22.	Boerma et al.	M	10	1	–	+	+	+	+	+	Mult.	Cured by lobectomy
23.	Burchell	M	17	12	–	+	+	+	+	–	Mult.	Cured by lobectomy
24.	Goldman	M	32	26	+	–	+	+	–	–	Sing.	Unknown
25.	Mallory et al.	F	23	7	–	–	+	+	–	–	Mult.	Cured by lobectomy
26.	Sweet	F	23	-	–	–	+	–	–	–	Sing.	No symptoms before and after lobectomy
27.	Sweet	M	2	1	–	–	+	–	–	–	Sing.	Cured by lobectomy
28.	Salvesen et al.	F	21	8	–	–	+	–	–	–	Sing.	Death by cause other than a.v.a.
29.	Salvesen et al.	M	37	25	–	+	+	–	–	–	Sing.	Cured by lobectomy. Death $2\frac{1}{2}$ years later by subarachnoidal hemorrhage
30.	Salvesen et al.	F	42	-	–	+	–	–	–	–	Sing.	Unknown
31.	Salvesen et al.	F	25	-	–	–	+	+	–	–	Sing.	Unchanged after 4 years
32.	Samson	M	25	-	–	–	+	–	–	–	Sing.	Cured by lobectomy
33.	Shefts	M	-	-	+	+	–	–	–	–	Sing.	Unknown

TABLE I (Continued)

Case	Author	Sex	Age	Age at onset of symptoms	Family history	Heman-giomas	Cyanosis and clubbing	Bruit	Nervous symptoms	Hemop-tysis	Number of aneurysms	Course
34.	Shefts Moyer et al.	M	25	15	+	+	+	–	+	–	Mult.	Cured by pneumonectomy
35.	Watson	M	27	-	–	–	–	–	–	–	Sing.	No symptoms before and after ligation
36.	Watson	M	21	19	–	+	+	–	+	–	Sing.	Cured by lobectomy
37.	Williams et al.	M	30	27	+	+	+	+	+	–	Sing.	Unknown
38.	Whitaker	F	44	1	+	+	+	–	–	–	Sing.	Death 6 days after lobectomy
39.	Whitaker	M	33	-	+	+	–	+	–	–	Sing.	Unknown. No symptoms
40.	Packard et al.	M	31	28	–	+	+	+	+	–	Mult.	Improved after ligation
41.	Barnes et al.	F	17	3	–	–	+	+	+	+	Sing.	Death 3 days after lobectomy
42.	Barnes et al.	M	42	26	–	+	+	–	–	+	Sing.	Improved after lobectomy
43.	Cleland	F	51	1	–	+	+	+	+	–	Mult.	Death 20 hours after operation
44.	Maier et al.	F	20	-	–	–	+	+	–	–	Sing.	Improved after lobectomy
45.	Moyer et al.	M	29	22	+	+	+	–	–	–	Sing.	Unknown
46.	Brobeck	F	34	-	–	+	+	+	–	–	Sing.	Cured by excision
47.*	Kunzer	M	6	1	–	+	+	–	+	–	Sing.	Unknown
48.	Giampalmo	M	21	7	–	+	+	–	+	–		Unknown

* Uncertain cases.

TABLE I (Continued)

Case	Author	Sex	Age	Age at onset of symptoms	Family history	Hemangiomas	Cyanosis and clubbing	Bruit	Nervous symptoms	Hemoptysis	Number of aneurysms	Course
49.	Wodehouse	M	21	-	–	–	–	–	–	–	Sing.	No symptoms before and after lobectomy
50.	Wodehouse	M	13	1	–	–	+	–	+	–	Sing.	Death by brain abscess
51.	Baer et al.	M	21	13	+	+	+	+	+	–	Mult.	Cured by lobectomy
52.	Blades	M	20	-	–	–	–	–	–	–	Mult.	No symptoms before and after lobectomy and excision
53.	Blades	M	22	21	+	+	+	+	+	–	Mult.	Cured by lobectomy and excision
54.	Yater et al.	F	42	41	–	+	+	+	+	–	Sing.	Cured by lobectomy
55.	Yater et al.	M	42	-	–	+	+	+	–	–	Sing.	Unknown. No symptoms
56.	Zanty	-	-	-	–	–	+	+	–	–	Sing.	Cured by pneumonectomy
57.	Hedvall	F	51	-	–	–	–	–	–	–	Sing.	Unchanged after 7 years
58.	Barker et al.	M	27	4	–	+	+	+	–	–	Mult.	Death 30 hours after lobectomy
59.	Barker et al.	M	13	5	–	–	+	+	–	–	Sing.	Cured by lobectomy
60.	Beddard	M	36	-	–	–	–	+	–	+	Sing.	Unknown
61.	Erf et al.	M	20	5	–	–	+	+	–	+	Sing.	Death by rupture
62.	Lawrence	F	30	24	–	–	–	+	–	–	Sing.	Improved after lobectomy
63.	Závodyn	F	60	-	–	+	–	–	–	–		Unknown
64.	Lequime et al.	F	65	20	–	+	+	–	+	–		Death by hemoptysis

TABLE I (Continued)

Case	Author	Sex	Age	Age at onset of symptoms	Family history	Heman-giomas	Cyanosis and clubbing	Bruit	Nervous symptoms	Hemop-tysis	Number of aneurysms	Course
65.	Garland et al.	M	6	3	+	+	+	+	–	–	Sing.	Cured by lobectomy
66.	Garland et al.	F	8	6	+	+	+	+	–	–	Sing.	Cured by excision
67.	Lawrence et al.	M	50	-	–	–	+	+	–	–	Sing.	No symptoms before and after lobectomy
68.	Duisenberg et al.	M	26	-	–	–	–	–	–	+	Sing.	Cured by lobectomy
69.	Carswell	M	20	-	–	–	+	–	–	–	Sing.	No symptoms before and after lobectomy
70.	Ettinger et al.	F	34	24	–	+	+	+	–	+	Sing.	Cured by lobectomy
71.	Robertson	M	27	-	–	–	+	+	–	–	Sing.	Death by pulmonary tuberculosis
72.	Friedlich et al.	M	15	5	–	+	+	–	–	–	Sing.	Cured by ligation
73.	Friedlich et al.	F	26	2	–	–	+	–	–	–	Sing.	Cured by lobectomy
74.	Friedlich et al.	F	8	7	–	–	+	–	–	–	Mult.	Cured by lobectomy
75.	Hedinger et al.	M	46	40	+	+	+	+	–	–	Mult.	Death by brain abscess
76.	Pugsley et al.	F	34	32	–	+	+	+	+	–	Mult.	Cured by excision
77.	Dubost et al.	-	-	-	–	–	+	–	–	–		Cured by lobectomy
78.	Nixon et al.	M	26	8	+	+	+	+	–	–	Sing.	Cured by pneumonectomy
79.	Muri, J.	M	49	-	–	–	+	+	+	–	Sing.	Death by brain abscess

conversly by the Müller test (expiration against closed glottis) the shadow will diminish. Sometimes, but not always, the shadow will pulsate. The proof of the vascular nature of the shadow may be given by angiography. The dye will concentrate in the aneurysm, and seldom will other aneurysms, not detected in the plain x-ray film, be visualized. The procedure is not without danger. Sisson et al., found these cases "more susceptible to untoward cardial effects because a larger amount of dye may go through the arteriovenous shunt to the left heart and the coronary vessels without having been through the mixing effect of the lung capillaries." After all it is a justifyable risk to take in order to get as full information as possible before surgical intervention. Nearly half of the patients have two or more aneurysms and often some of these remain undetected until the angiography.

The nomenclature is somewhat confused. Often the lesions are named hemangiomas or cavernous hemangiomas of the lung. Adams et al. state that "on the basis of the pathologic picture, the lesions are really arteriovenous aneurysms or fistulas." Lindgren proposes the name aneurysm for congenitial lesions, reserving the term fistula for the (rare) traumatic shunts. Hemangiomas of the lung are tumors with a quite different clinical and pathological aspect. (37).

The diagnosis is based on the history of the patient and that of the family. Cyanosis and clubbing of the fingers and toes and secondary polycythemia will raise the suspicion of a right-to-left shunt. Normal heart sounds and, if present, a systolic murmur over the lungs will make the diagnosis probable. Other causes of polycythemia must be ruled out. In polycythemia vera the white cell count is high with a shift to the left. The blood pressure is usually elevated, the spleen may be palpable and the patient looks more red than blue. Further blood studies will make out this diagnosis. Pulmonary diseases preventing adequate oxygenation, such as fibrosis and emphysema, will rarely cause cyanosis so pronounced as a right-to-left shunt in the heart or the lungs. Radiographic examination of the chest will be decisive in these cases. Many of the patients have had a previous diagnosis of tuberculosis because of hemoptysis and a shadow in the lung. In only one of the reported cases this disease coexisted with an pulmonary arterio-venous aneurysm (49). X-ray findings are important not only by excluding cardiac malformations and lesions in the lungs other than pulmonary arterio-venous aneurysm, but also by the characteristic feature of the aneurysm. Cyanosis caused by poisoning with heavy metals, aniline dyes, etc., may be excluded by the history. Ultimately the diagnosis may be confirmed by angiography.

Of the 79 cases in the table, the diagnosis is probable in 4 (x). In six the diagnosis was made post mortem. In one (17) the cause of death was not mentioned. In eight the cause was rupture of the aneurysmal sack into a bronchus or in the pleural cavity. In one case the autopsy revealed an abscess in the brain. This was the cause of death in three additional cases where the diagnosis was made ante mortem. It seems probable that these

patients are less resistent to infections than normal. In many reports mention is made of osteomyelitis, bronchitis and abscesses elsewhere.

Of the 31 cases which were not operated, four died from causes not related to the pulmonary lesion. Follow-up examinations are available in only a few cases. Hedvall's case remained stationary during seven years. In one of Salvesens cases the lesion was stationary for four years. In the case of Makler and Zion the clinical picture was unchanged after four years, but the hemoglobin value had increased from 19.5 Gm. in 1946 to 22.3 Gm. in 1950 (22) when he was operated on with a good immediate result. Expectancy seems fairly good when cyanosis is absent or slight, but when cyanosis has developed and polycytemia is pronounced, progression will most often be rapid.

Forty-eight cases were operated with a primary mortality of 8.3 per cent. One patient died during angiography with 70 per cent diodrast (56). In three cases ligature of the "feeder artery" was performed (22, 46, 59) with improvement in two and good result in one where the artery came from the aorta. In the other cases the lesion was removed by segmental resection, lobectomy or pneumonectomy. Most often the immediate result is excellent with disappearance of cyanosis in a few hours, and a return to the normal of the blood values in some weeks. In some cases there remains a certain degree of arterial unsaturation which may be caused by additional not diagnosed aneurysms. Late results are not available for obvious causes.

In making a decision whether to operate, it is necessary not only to consider the number and site of the lesions, but also the natural course and the actual state of the disease and the age of the patient. Most of the patients are in the third decade, and ought to be operated if there are cyanosis and clubbing and/or alarming nervous symptoms or hemoptysis.

SUMMARY

1) In a 48 year old male with cyanosis and clubbing of the fingers and a murmur over the base of the left lung the diagnosis of an arteriovenous aneurysm was made clinically. The diagnosis was confirmed by x-ray examination and planigraphy which showed a vascular loop in the left lower lobe. The patient died of brain abscess and the aneurysm was demonstrated by injection with polyvenylacetic esters.

2) A review is given of the cases reported to-date (Table I) with their chief clinical characteristics. Cases without cyanosis may be explained by the existence of a fistula between a bronchial artery and a pulmonary vein.

3) The most serious complication is rupture of the aneurysmal sack into a bronchus or into the pleural space. Neurological symptoms are common. These patients seem to have poor resistance against infections. Four of the cases died of brain abscess.

4) In spite of the characteristic clinical picture, x-ray examination is of the utmost importance for the diagnosis, and before surgical intervention angiography ought to be done because of the frequent multiplicity of the lesion.

RESUMEN

1) En un hombre de 48 años con cianosis y dedos hipocráticos asi como un soplo en la base del pulmón izquierdo, se hizo el diagnóstico de aneurisma arteriovenoso clinicamente. El diagnóstico fué confirmado por el exámen a los rayos X y por la planigrafía que mostró un arco vascular en el lóbulo inferior izquierdo. El enfermo murió de absceso cerebral y el aneurisma fué demostrado por la inyección de esteres de polivinilacético.

2) Se revisan los casos hasta ahora relatados (Cuadro 1) con sus principales caracteristicas clinicas. Los casos sin cianosis pueden explicarse por la existencia de fistula entre la arteria bronquial y una vena pulmonar.

3) La complicación mas seria es la ruptura del saco aneurismal dentro del bronquio o dentro del espacio pleural. Los sintomas neurológicos son comunes. Parece que estos enfermos presentan escasa resistencia frente a las infecciones. Cuatro de los casos murieron de abscesos cerebrales.

4) A pesar del aspecto clinico caracteristico el examen a los rayos X es de la mayor importancia para el diagnóstico y antes de la intervención quirúrgica la angiografía debe hacerse a causa de frecuente multiplicidad de las lesiones.

RESUME

1) Chez un homme de 48 ans, atteint de cyanose, d'hippocratisme digital, et d'un souffle de la base du poumon gauche, on posa le diagnostic clinique d'anévrysme artério-veineux. Ce diagnostic fut confirmé par les examens radiographiques et tomographiques, qui montrèrent une boucle vasculaire dans le lobe inférieur gauche. Le malade mourut d'un abcèss du cerveau, et la démonstration de l'anévrysme put se faire par injection d'éther polyvenylacétique.

2) L'auteur passe en revue des cas qui ont été rapportés jusqu'à présent, avec leur principales caractéristiques cliniques. Les cas qui se passent de cyanose peuvent être expliqués par l'existence d'une fistule unissant une artère bronchique à une veine pulmonaire.

3) La plus sérieuse complication est la rupture du sac anévrysmal soit dans une bronche, soit dans l'espact intra-pleural. Les symptomes neurologiques sont fréquents. Ces malades semblent être très peu résistants aux infections. Dans quatre observations, la mort survint par abcès du cerveau.

4) Malgré l'allure clinique caractéristique, l'examen radiologique est d'importance capitale pour le diagnostic, et avant l'intervention chirurgicale, il est nécessaire de faire une angiographie étant donné la fréquence des lésions multiples.

Streptomycin in Silicotuberculosis*

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Theodos and Gordon state that the life expectancy in silicosis with complicating tuberculosis treated at home is two or three years, and that with sanatorium care life expectancy in some cases may be increased to four years.[1] If the disease is clinically inactive, no particular treatment is necessary, and the patients may be encouraged to continue at work; they must in such cases work in an atmosphere which is free of dust. However, when tuberculous cavitation exists and the sputum or gastric contents are positive for tubercle bacilli, the disease usually progresses relentlessly to a fatal termination. Isolation is advisable to prevent infection of others, and rest may be of some help. Collapse therapy is usually impractical or impossible;[1-3] silicosis itself reduces the vital capacity and may cause dyspnoea; extensive pleural adhesions prevent adequate collapse; and tuberculous cavities in silicotic areas of the lung are difficult to close by any collapse measures. Auerbach and Stemmerman reviewed eight cases of silicotuberculosis in which artificial pneumothorax was attempted, and four in which thoracoplasty was performed; in none of these cases was a cavity closed.[4] They believe that there is little hope for success with collapse therapy in silicotuberculosis. The silicotic areas form a resistant barrier to collapse of lung tissue. In ordinary tuberculosis there is destruction of the elastic elements of the lung, and when collapse therapy is attempted it may readily be successful, as the pulmonary parenchyma has already suffered a decrease in volume. In silicosis, on the other hand, the fibrosis occurs chiefly along the septa, the silica particles stimulating the formation of new collagen fibrils to form connective tissue. The silicotic lung is more voluminous than normal and shows no tendency to collapse. Some authors even believe that in advanced cases life is actually shortened by collapse therapy.[5] Because of widespread disease and lowered vital capacity, resection of major tuberculous foci is seldom feasible.

Because of the poor results obtained with other forms of treatment, streptomycin was given to every patient who came to this hospital with advanced tuberculosis complicating silicosis. For purposes of this study, no case was accepted unless there was a good history of prolonged exposure to silica dust, an x-ray film appearance typical of nodular or conglomerate silicosis and cavitation and sputum positive for acid-fast bacilli. It is admitted that this is the type of disease in which in the past treatment

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has been almost uniformly unsuccessful. Eighteen such cases were treated. The results were poor.

Symptomatic improvement—a feeling of well-being, improved appetite, weight gain, and decrease of fever—was seen in only three cases, and significant improvement was demonstrated by x-ray film inspection in only one. In several other cases there was slight and temporary improvement by x-ray film inspection. Review of these 18 cases 12 months after treatment was started, showed that six were dead, seven were worse, four were unchanged, and only one was improved. In not a single instance was there cavity closure or sputum conversion; in not a single instance did collapse or excision become feasible as a result of streptomycin therapy.

TABLE 1

RESULTS ONE YEAR AFTER START OF CHEMOTHERAPY

Number of cases	18
Dead	6
Worse (x-ray)	7
Unchanged (x-ray)	4
Improved (x-ray)	1
Improved (symptoms)	3
Sputum conversion	0

The only patient who showed significant improvement was a 40 year old white man who had worked for nine years in an anthracite mine in Eastern Pennsylvania. Chest x-ray film in 1947 showed a nodular pneumonoconiosis throughout, with large conglomerate nodules in both upper lobes. He was not allowed to return to the mines, but went to work as a floor layer. He remained asymptomatic except for slight dyspnoea until November 1950, when he developed some cough and expectoration. Thereafter these symptoms progressed and he lost weight. Chest x-ray film showed evidence of a new lesion at the right base, with a large cavity, and sputum was positive for acid-fast bacilli. On treatment with streptomycin his appetite improved

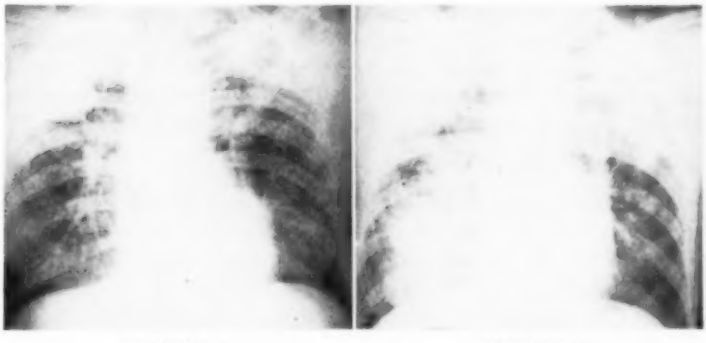

FIGURE 1 FIGURE 2

Figure 1: Extensive Conglomerate Silicosis.

Figure 2: Tuberculous lesion with cavitation in right lower lobe, in same patient.

and he gained 14 pounds in weight. Simultaneously serial x-ray films showed moderate improvement in the tuberculous lesion, but the cavity remained open, the sputum remained positive for acid-fast bacilli on concentration and culture, and was repeatedly heavily tinged with blood. Further improvement is not occurring on a medical regimen, and the thoracic surgeons who have seen the patient show no enthusiasm to accept the case for collapse or excision. It should be repeated that this is a report of our most successful case.

There are occasional reports in the literature which indicate that streptomycin is of value in silicotuberculosis. In most of these reports either the silicosis or the tuberculosis are not extensive. Boselli and Lusardi[6] gave streptomycin to 23 cases with the combined diseases, and assert that in every case the treatment brought about benefit. Most of their patients had no pulmonary cavities. They note that on chemotherapy, in spite of clinical improvement, no cavities closed, and they say nothing about sputum conversion in cavitary cases. Most observers are agreed that in chronic fibro-cavitary silicotuberculosis streptomycin is of little value,[2,7] except to reduce the manifestations of toxemia,[1] and the results in our cases certainly support this view.

If streptomycin is of value in silicotuberculosis, its benefit is limited to acute progression of tuberculosis, post-hemorrhagic spreads, miliary disease, and extra-pulmonary tuberculosis. It is probable that before cavitation develops streptomycin may be helpful. In chronic cases with cavitation and positive sputum streptomycin does not appear to cause improvement or even halt the progression of the tuberculous disease.

SUMMARY

The very hopelessness of the treatment of extensive fibro-cavitary silicotuberculosis brings with it hope for the eradication of this disease. Since it is impossible to treat successfully this combination of diseases in its late stages, it must be detected earlier when treatment may be of value. This calls for periodic examinations of silicotics, alertness for early evidence of tuberculosis, and treatment at this stage. Better yet, since both silicosis and tuberculosis are preventable diseases, and since much progress has already been made in their prevention, it seems only a matter of time until silicotuberculosis will cease to exist and its treatment will be merely of academic interest.

RESUMEN

La falta de esperanza en el tratamiento de la tuberculo-silicosis extensiva y cavitaria, trae consigo la esperanza de que la tuberculosis sea erradicada. Puesto que es imposible tratar con exito esta combinación de enfermedades en sus etapas tardías, debe ser descubierta más tempranamente, cuando el tratamiento puede ser de valor. Esto requiere exámen periódico de los silicosos, estar alerta para cualquier evidencia de tuberculosis y tratamiento en esa etapa. Mejor aún, puesto que tanto la silicosis como la tuberculosis pueden prevenirse, parece que es solo cuestión de tiempo el

que la silico-tuberculosis desaparezca y entonces su tratamiento sea solo algo de interés académico.

RESUME

L'insuccès complet du traitement de la silico-tuberculose extensive avec lésions fibro-ulcéreuses ne fait entrevoir d'espoir que dans la suppression de cette maladie. Puis-qu'il est impossible de traiter avec succès cette maladie à sa phase tardive, on doit la déceler plus tôt, lorsque le traitement peut avoir quelque effet. Ceci demande l'examen systématique des silicotiques, afin de mettre en évidence la tuberculose quand elle est encore latente, et de permettre son traitement. Mieux encore, puisque silicose et tuberculose sont des affections qu'on peut éviter, et que beaucoup de progrès ont été réalisés pour leur prévention, il semble que ce soit seulement une question de temps pour que la silico-tuberculose cesse d'exister et que son traitement ne soit plus que d'un intérêt purement théorique.

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Esophageal Fistulae Complicated by Mycotic Empyema and Esophago-Aortic Perforation

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Abnormal esophageal communications may assume various forms, depending upon the direction in which they point and what contiguous structures lie in their paths. Thus, there may be communications between the esophagus and skin, pleura, trachea, bronchi, pericardium, and large vessels.

In order of frequency, the most common causes of perforation of the esophagus are carcinomas, congenital deformities, infections, and trauma. The process may begin extrinsically as a periesophagitis, from the caseation and ulceration of a tuberculous lymph node, from a primary or metastatic periesophageal malignancy, or from an empyema necessitatis rupturing into the esophagus.[1-4] Simultaneous perforation of the esophagus and trachea from rupture of an aortic aneurysm has occurred, and a case has been reported of a fistula arising from a cold abscess of the vertebral column.[2] The intrinsic lesions include carcinoma of the esophagus, tuberculosis, syphilis, mycosis, acute esophagitis, infected diverticula particularly of the traction type, simple ulcers, cicatricial stenoses, foreign bodies, infarction, cardiospasm, and spontaneous rupture in association with vomiting, purgation, and severe sepsis.[1-3,5-8] When a perforation of a great vessel has occurred, the cause most frequently has been the trauma incident to a foreign body within the esophagus.[9-13]

The following case is of interest because of the simultaneous presence of an esophago-pleural communication aggravated by a monilial infection to produce severe mycotic tension pyopneumothorax and a concomitant esophago-aortic fistula.

Case Report

A.G., No. 214966, a 52 year old white woman was admitted to the Bronx Hospital on June 19, 1949 with a chief complaint of dyspnea and weakness. She had been feeling irritable and had suffered pain in her right chest posteriorly for the past few weeks. The pain was not of sufficient severity, however, to disturb her daily routine as a saleslady in a bakery shop. On May 26, 1949, she experienced a sticking pain in the right side of the chest posteriorly which penetrated through to the sternum. She felt very weak and had to remain in bed. At this time, she could not tolerate solid food and maintained herself on liquids only. On May 29, she had severe knifelike pains in her back and right shoulder and developed some abdominal distention. It was also discovered that she was diabetic and she was placed on insulin. Back pain, fatigue, weakness, and fever persisted and although hospitalization was advised at this time, it was refused by the patient. She was therefore maintained at home with sedation and given penicillin and streptomycin. On June 2, because of increasing dyspnea, oxygen was given at home. The next day she felt better. She had more strength and less pain. While moving about in bed, she

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coughed, producing a small amount of bloody sputum. On June 8, a right thoracentesis was attempted by the family physician, but no fluid was obtained. The following day she was admitted to the Bronx Hospital.

Past history and family history were essentially negative.

Inquiry by systems revealed that she had slight shortness of breath for several years. There had been no ankle edema, precordial pain, or nocturnal dyspnea. There was no history of previous cough, hemoptysis, night sweats, fever, or chest pain prior to the present illness. She had always had a thirst for carbonated beverages which became more intense in the few months prior to admission. Her appetite was normal. On occasion, however, she had a sense of oppression substernally, particularly when eating, with a feeling that food did not pass down the esophagus as it should. Her bowel movements were regular. She had no nausea, vomiting, food intolerance, or abdominal pain. She had never noticed bloody or tarry stools. The patient had always voided large quantities of urine at frequent intervals. There was no nocturia, pain or burning on urination, hematuria, or pyuria. Menopause occurred at 44 years of age. Prior to this, her periods were regular.

On admission, she appeared acutely ill. She was restless, dyspneic, orthopneic, and slightly cyanotic. Her temperature was 100.4 degrees F., pulse rate 110, and respirations 32 per minute. There was dullness over the lower half of the right chest. The hemoglobin was 60 per cent, red blood count 3,000,000, white blood count 25,000, neutrophils 85 per cent, stab forms 4 per cent, juvenile forms 1 per

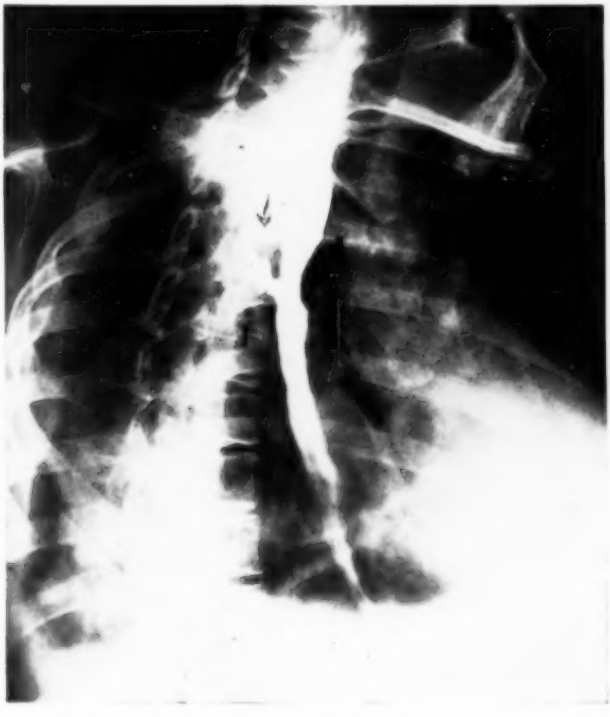

FIGURE 1: Right anterior oblique view of thorax and esophagram illustrating two esophageal fistulae.

FIGURE 2

FIGURE 3

Figure 2: Opened esophagus, lungs, and mediastinal structures with probes extending, through fistulous openings. (**A**) Penetrated through into aorta. (**B**) Communicated with the right pleural cavity.—*Figure 3:* Fistulous opening into aorta.

cent, lymphocytes 6 per cent, monocytes 4 per cent, normoblasts 2 per cent. The urinary sugar varied from 0 to 3 per cent. There was no acetone.

It was our impression that the patient had an acute pyothorax which was confirmed by inserting a needle through the right anterolateral chest wall, resulting in the immediate evacuation of pus under tension. An open thoracotomy was performed under local anesthesia, resecting segments of the eighth and ninth rib in the right midaxillary line. On entering the pleural cavity, there was an immediate evacuation of yellowish-gray pus containing numerous whitish flakes. The material had a foul, acrid odor. Three drains were inserted and the wound packed around the drains to institute open drainage. Immediately following the thoracotomy the dyspnea improved considerably, and by the time she was returned to her room she was fairly comfortable. Examination of the material removed from the chest at operation revealed a fungus-like organism in pure culture. This was identified as Monilia albicans. The finding of a pure mycotic empyema was unusual and we postulated at this time the possibility of esophageal perforation.

Postoperatively, she was given penicillin and streptomycin. For the first five days, there was a moderate amount of grayish-yellow discharge from the wound, the temperature remained around 100 degrees F., and there was little respiratory distress. On the fifth postoperative day, she developed a large red vesiculated eruption surrounding the thoracotomy wound. This was interpreted as a sensitivity reaction to penicillin and streptomycin. These were discontinued and mild Dakin's solution irrigation of the chest was commenced. A culture of the material draining from the chest now revealed B. proteus, E. coli, and yeast-like cells. Blood culture was negative. With discontinuance of penicillin and streptomycin the vesiculated skin eruption cleared up promptly. She was then placed on increasing desensitizing doses of these antibiotics and the former dosage was rapidly resumed. On June 16, potassium iodide was started by mouth in doses of 15M t.i.d.

On June 23, an esophogram was done which revealed two irregular penetrations into the mediastinum above the bifurcation of the trachea. The appearance was not that usually seen with traction diverticula, but one suggesting rather irregular sinus formations.

On June 27, the 17th postoperative day, she suddenly had hematemesis of 50 to 100 cc. of blood followed by repeated emesis of dilute bloody material. She went into shock, exhibiting a cold clammy skin, rapid pulse, and drop in blood pressure. She had no respiratory distress, but complained of vague abdominal pain and a sense of oppression. Her abdomen was soft, and there was no localized area of tenderness. She responded quickly to a transfusion of 500 cc. of citrated blood. The next day she felt well. There was no further substernal distress or abdominal pain. That evening, however, while getting off a bed pan, she had a sudden emesis of a moderate amount of bright red blood. She rapidly went into shock and died despite all supportive measures.

Necropsy Observations (Pertinent data):

Gross: There was marked pallor of all the viscera. The stomach was dilated and contained several large blood clots which completely filled the lumen. The stomach itself showed no gross pathologic change. In the esophagus, at the level of the arch of the aorta, there were two cleanly punched-out fistulae, 3 cm. in diameter. The one on the right side opened into the right pleural cavity by a tract that could be probed. The one on the left opened into the aorta. Within the latter fistula was a small amount of pink tissue. No foreign body was found in this area and there was no gross inflammatory reaction of the mucosa of the esophagus. There was no free pleural fluid. There were extensive adhesions between the right lung and all surrounding structures. The visceral pleura was markedly thickened. On section there was a localized collection of greenish-yellow pus (10 cc.) in the tissue between the right middle and right lower lobes. The rest of the lung was grossly normal. There was no remarkable hilar lymphadenopathy. Smears of the inter-

lobar empyema revealed numerous leucocytes, occasional gram-positive, coccus-like shapes, and mycelial threads.

Microscopic: One section of the esophagus showed an area of chronic inflammation in the mucosa with slight extension into the muscularis. In the involved area there was considerable congestion, edema, and infiltration by many round cells and occasional histiocytes, multinucleated giant cells, and polymorphonuclear leucocytes. Another section demonstrated the communication between the aorta and esophagus at the point of rupture of both of these structures. Lining the cavity between these two structures was an adherent blood clot. At the point of rupture both the esophagus and aorta showed thinning out, degeneration, and chronic inflammation. The aorta was partially hyalinized in that area and the esophagus showed thinning out of the mucosal coat with atrophy of the epithelium. There were many more polymorphonuclear leucocytes in the exudate in this section than in the previous one.

Two sections of lung showed a similar picture. The parenchyma was the seat of moderate congestion with areas of emphysema and atelectasis, although most of the parenchyma was well aerated. The pleura was markedly thickened due to extensive fibrosis. The connective tissue was infiltrated by many polymorphonuclear leucocytes, round cells, histiocytes, and occasional multinucleated giant cells. Another section of lung showed pulmonary edema and congestion.

Comment

The exact etiology of the esophageal fistulae in this case could not be definitely established from the history or autopsy findings. There was no specific intrinsic or extrinsic lesion directly responsible. It is our feeling that trauma may have been the initiating factor with subsequent superimposed monilial infection. Monilial infections occur frequently in the mouths of diabetics because of the high carbohydrate concentrations of the body secretions.[14] The finding of a pure monilial infection of the pleural space probably resulted from the suppression by antibiotics of other organisms that are usually present in the mouth and esophagus.

Acknowledgement: We wish to express our thanks to the Department of Pathology, Dr. Joseph Felsen, Director, Bronx Hospital, New York City, for their cooperation and assistance.

SUMMARY

A case is reported with multiple fistulization of the esophagus resulting in an esophagopleural communication, a mycotic empyema, and esophagoaortic perforation. Trauma with superimposed infection was probably the predominant factor in the etiology of this condition.

RESUMEN

Se presenta un caso con fistulización múltiple del esófago resultante de una comunicación esófago-pleural, un empiema micótico, y una perforación esófago-aortica. Probablemente el trauma, agregado a infección, fué el factor predominante en la etiología de esta condición.

RESUME

Les auteurs rapportent un cas de fistules multiples de l'oesophage, consécutives à une communication oesophago-pleurale, une pleurésie purulente

d'origine mycosique, et une perforation oesophago-aortique. Cet état à vraisemblablement pour cause essentielle un traumatisme auquel s'est surajoutée une infection.

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The Use of Beta-Glucuronidase as a Measure of Pleural Irritation*

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It has been shown that the enzyme Beta-Glucuronidase is present in the fluid which develops in the pleural cavity during certain diseases.[1] Whether this enzyme occurs as a result of migration through the pleura from the blood, or whether it is secreted by the epithelium lining the pleural cavity has not been determined.

In our studies at this hospital on pleural effusions, we have assayed Beta-Glucuronidase activity on the fluid before and after administration of therapeutic agents, in order to determine the effect that the drug has had on the enzyme level. The substances used were:

(1) Streptokinase-Streptodornase (SK-SD)
(2) Trypsin (Tryptar)
(3) Hyaluronidase
(4) Streptomycin
(5) Penicillin
(6) Para-aminosalicylic Acid (PAS)
(7) Methylene blue.

In a few cases with spontaneous bleeding the irritative effect of blood was also studied.

Method

Patients with pleural effusions in whom therapy seemed indicated were aspirated and the pleural cavity was drained as nearly as possible. In some instances the air from a pre-existing pneumothorax was present. No special attempt was made to aspirate this. The desired therapeutic agent, in solution, was then injected through the aspiration needle, and the needle was removed. Unless otherwise noted, the fluid was re-aspirated after 24 hours and again the pleural cavity was, as nearly as possible, drained dry. For computation purposes, most of the patients were fluoroscoped after aspiration and an estimate was made of the volume of fluid which remained in the chest after aspiration.

The Beta-Glucuronidase activity of the fluids before and after administration of the medicament was determined. The phenolphthalein glucuronidase method of Fishman, et al.,[2] was used except that the filtration was omitted. It was found that by deletion of this step, the procedure was simplified with sacrifice of a minimum of accuracy. All assays were run in duplicate. The greatest variation was 8 per cent; mean variation, 2 per cent. The amount of phenolphthalein liberated was determined colorimetrically.

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Results

(1) *Trypsin (Tryptar):* One vial, containing 250 mg. and diluted to 25 cc. with Sorensen's phosphate buffer solution was injected into the chest cavity, after removing as much fluid as possible. In most cases, this was not attended by a febrile or toxic reaction. After 24 hours, the amount of fluid had usually returned to, or exceeded, its original level. It had assumed a pinkish tinge and, in most cases, was more turbid than the original fluid.

The Beta-Glucuronidase activity was increased in all cases (See Table I).

(2) *Streptokinase-Streptodornase (SK-SD):* The pleural reaction to these enzymes was similar to that following tryptar, except that most patients suffered toxic reactions (fever, chills, anorexia, etc.). The chest fluid also, in addition to gaining a pinkish tinge, seemed to acquire an opalescence which remained even after centrifugation. The method of administration was similar to that of tryptar, one therapeutic vial containing 100,000 units SK and 25,000 units SD being used in each case. There was an increase in Beta-Glucuronidase activity in each case (Table II).

Opportunity for observing the effect of the other agents was small be-

TABLE I

This table shows the estimated total amount of fluid present in the pleural cavity before aspiration (i.e., volume of fluid removed and amount estimated, fluoroscopically, to remain), the Beta-Glucuronidase activity (ABG) of that fluid, the estimated volume approximately 24 hours after instillation of tryptar, and the activity of the fluid at this time. Those values followed by (+) were clear.

	BEFORE TRYPTAR		24 HOURS AFTER TRYPTAR	
	Volume	AB-G	Volume	AB-G
1.	500	738 +	700	1143 +
2.	550	1091 +	600	1350
3.	550*	1000	600*	1666
4.	200	3090	200	6000
5.	200*	2700	200	3428
6.	800	333 +	1000	1517 (48 hrs.) +
7.	300	180 +	300	1280

*Volume removed. Patient was not fluoroscoped after aspiration.

TABLE II

This table shows the estimated volume of fluid and Beta-Glucuronidase activity before and 24 hours after treatment. (+)=Clear fluids.

	BEFORE SK-SD		AFTER SK-SD	
	Volume	BAG	Volume	BAG
1.	1700	1142 +	1700	2500
2.	800	1412	300*	2222
3.	1000	1620	1200	2000

*Volume removed. Fluoroscopy was not done.

cause of infrequent use; however, the results that we have are summarized in Table III. Again in each case the activity was increased.

Moderate bleeding occurred during a pneumonolysis operation in one patient who had had a minimal amount of fluid, which had been assayed previously. This gave us an opportunity to study the irritative effect of blood in pleural fluid. The bloody fluid was aspirated 24 hours after the operation and the enzyme level determined. It was found to be significantly higher than either the earlier level in the pleural fluid or the level in the circulating blood.

Discussion

An increase in the Beta-Glucuronidase activity of pleural fluids was noted after the addition of each of a number of intrapleural therapeutic agents. In a few cases, the fluids were classified as clear, both before and after the administration of the medicant.

These irritants were therapeutic agents normally employed in the therapy of chest conditions and included streptokinase-streptodornase, trypsin, hyaluronidase, etc. The relative amount of irritation which any one of these might produce is unknown. It would seem, however, that inasmuch as the enzyme level in every case was increased, perhaps this increase in Beta-Glucuronidase level reflects the irritative effect of the medicant.

In most instances the fluid was as turbid, or more so, following administration of the therapeutic agent, than prior to therapy. Inasmuch as turbidity was estimated, it is possible that the increased activity is due to the presence of a greater number of leucocytes in the fluid.

A few of the fluids were clear both before and after therapy. On centrifugation only a minimal amount of sediment could be seen. The fact that high concentration of the enzyme can sometimes be found in clear pleural fluids has been noted previously.[1] If this fact is to be explained on another basis, it must be that the pleural wall itself secretes the enzyme when irritated.

TABLE III

This table shows that several foreign agents in the pleural cavity tended to cause an increase in the amount of fluid as well as to increase the Beta-Glucuronidase activity. The column ABG represents the Beta-Glucuronidase activity.

	Before Treatment		24 Hours After Treatment	
	Volume	ABG	Volume	ABG
1. Streptomycin	300	1200	300	2050
2. Penicillin	1000	250 +	1200	350 +
3. PAS	300	1250	400	1350
4. Hyaluronidase	500	450 +	500	1250 +
5. Methylene blue (1)		3150		3542*
(2)		2311		5000
6. Blood	200	1050 +	1200	1250 (bloody)

*Both cloudy fluids. Volumes not available. + Clear fluids.

The pleural membrane has been shown to be an ineffective barrier toward moderate-sized chemical molecules.[3] The high protein levels in pleural effusions would lead us to believe that these also may pass easily through it. Following physical laws, then, high levels of Beta-Glucuronidase secreted into the pleural cavity would be expected to be re-absorbed in a short time into the blood stream.

It is interesting to speculate on the possibility that the relative toxicity of any therapeutic agent on tissue would be a function of its effect on the Beta-Glucuronidase activity of a pleural effusion. If this were the case, in order to compute accurately the amount of irritation which any one agent produces, several factors would have to be considered, namely, the area of pleura in contact with the irritant, the amount of fluid produced, the concentration of the irritant injected, and the length of time elapsed since the injection. An attempt is being made to further clarify this by animal experimentation.

SUMMARY

1) The effect of a number of intra-pleural therapeutic agents increasing the Beta-Glucuronidase activity of pleural fluids is presented.

2) Evidence that the pleural epithelium when irritated acts as a differentially secretory organ for the enzyme is discussed.

RESUMEN

1) Se presenta el efecto de ciertos agentes terapéuticos que aumentan la actividad de la Beta-Glucoronidasa de los líquidos pleurales.

2) Se discute la evidencia de que el epitelio pleural cuando es irritado actua como un órgano diferenciado secretorio de la enzima.

RESUME

1) L'auteur étudie l'action d'un certain nombre d'agents thérapeutiques par voie intra-pleurale, qui augmentent l'activité de la Beta-Glucuronidase des épanchements pleuraux.

2) Il discute l'affirmation que l'épithélium pleural, lorsqu'il est irrité, se comporte pour l'enzyme comme un organe différencié.

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Effects of Intravenously Administered ACTH on the Pulmonary Function in Bronchial Asthma and Emphysema*

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The usefulness of ACTH and cortisone in the treatment of bronchial asthma is now well established. Many reports have shown that the signs and symptoms of this disease are markedly alleviated by treatment with these hormones.[1-5] In a number of cases this treatment has been proved to be the only satisfactory means for the remission of an intractable attack of asthma.

Most cases of chronic bronchial asthma are accompanied by signs of obstructive emphysema.[6,7] Lung function studies may therefore be important in the evaluation of the clinical condition and the effects of treatment. Reports on such investigations after treatment with ACTH and cortisone are few. Galdstone et al.[8] reported studies in two cases of bronchial asthma, emphysema and fibrosis. One of these patients also had chronic cor pulmonale. The treatment with ACTH caused marked improvement in the clinical course and in the lung functions in one case, while in the case with cor pulmonale no improvement could be obtained. Lukas[9] reported nine patients with bronchial asthma and chronic obstructive emphysema, but included in this series one case of sarcoidosis, one of scleroderma and one of bullous emphysema. Clinically, all experienced some improvement following administration of ACTH. In all cases there was an increase in the maximal breathing capacity (MBC). In six there was also a rise in vital capacity (VC), although the degree of this rise was usually less marked than that of the MBC; in the remaining three cases the vital capacity even decreased. A more detailed study of pulmonary functions in four cases revealed, as the most significant result, a decrease in the residual volume which proves a reduction of the emphysematous state. In one of the four cases, who was suffering from cor pulmonale, the arterial oxygen saturation, which was already low before treatment, decreased still further. This finding indicates a deterioration of the respiratory function of the lungs. The development of interstitial edema of the alveoli was thought to be responsible for this deterioration.

In the following we are reporting the effects of intravenously administered ACTH on pulmonary functions in patients with bronchial asthma and emphysema. Although our studies were concerned mainly with the ventilatory function of the lungs, we thought it worthwhile to report them, in the belief that every additional study may help in the proper selection of patients for future hormonal therapy.

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Material and Methods:

Nine patients, ranging in age from 24 to 65 years, were studied. All of them had a clinical diagnosis of long standing bronchial asthma and emphysema. The usual manifestations, such as wheezing, rhonchi sonores and sibilantes, were present. None showed signs of congestive heart failure. During the course of treatment no therapy other than emergency drugs was given. Two to 5 mgm. of ACTH were administered daily by continuous intravenous infusion in 500 to 1,000 ml. of a solution of 5 per cent glucose with the addition of 0.2 per cent potassium chloride.[10] The rate of the drip was from 12 to 16 drops per minute. This treatment was given over a period of from five to 10 days.

Lung function determinations: All the studies were carried out under basic conditions before and following ACTH administration. For all the measurements a Collins respirometer with an attachment for the measurement of the ventilation was used, and lung volume determinations are expressed at a temperature of 37 degrees C., ambient pressure, saturated. The maximal breathing capacity (MBC) was measured by the method of Hermannsen as recently described by Cournand and Richards.[11] Maximal breathing capacity and vital capacity were recorded in the erect position. The per cent of the predicted maximal breathing capacity and the predicted vital capacity was calculated according to formulae recently described by Baldwin et al.[12] The fraction $\frac{\text{Per cent of predicted MBC}}{\text{per cent predicted VC}}$ which has been designated by Gaensler[13] "air velocity index," was determined for every patient before and after treatment. (The air velocity index is less than one if the ventilatory insufficiency is due to increased resistance in the air passages; the index is one or higher in normals and in patients with stroke volume defects). The breathing reserve, i.e. the difference between the maximal breathing capacity and the minute volume of breathing, was calculated. The breathing reserve ratio, which is $\frac{\text{breathing reserve}}{\text{MBC}} \times 100$, was estimated. (The normal value for the breathing reserve ratio of resting subjects is 95.9 per cent, and according to some authors dyspnea is experienced when values are less than 65 per cent). The oxygen removal per litre of ventilation was calculated from spirographic tracings.

Results:

All patients, with the exception of one (N.B.) showed clinical improvement during the treatment. They were relieved from asthmatic attacks or from status asthmaticus, from which they had been suffering for some time prior to treatment. It has been found that the small doses given intravenously by drip were at least as effective as the larger doses administered intramuscularly.

As shown in Table I, the first five cases showed a similar response to the treatment. The maximal breathing capacity increased by five to 19 per cent of the predicted values. The breathing reserve rose by seven to 24

litres per minute. Accordingly, the breathing reserve ratio also rose by two to 16 per cent (Figure 1). The vital capacity rose by six to 29 per cent of the predicted values. The oxygen removal per litre ventilation, which is one of the indicators of the gas exchange in the alveoli, also showed improvement in all these cases. In four cases of this group the air velocity index was found before treatment to be less than one. The marked prolongation of the expiratory slope in the vital capacity tracings, which was noted before treatment, diminished markedly after therapy.

The four other cases (R.F., S.B., N.B., G.M.) did not show a uniform response in the lung function tests. Some of the functions improved but most of them deteriorated. Thus the breathing reserve ratio in all these cases decreased.

Comment

The maximal breathing capacity and the vital capacity were reduced in all cases before treatment with ACTH. These findings may indicate that the ventilatory insufficiency was due to obstruction of the air passages, as well as to a diminution of the stroke volume. However, it should be noted that in four cases out of the five which showed improvement in the pulmonary function after ACTH treatment, the air velocity index was less than one, which proves that the maximal breathing capacity was reduced out of proportion to the decrease in the vital capacity. It may, therefore, be assumed that the predominant factor in the causation of

TABLE I

THE EFFECT OF INTRAVENOUSLY ADMINISTERED ACTH

	J.H. M-27 yrs.		R.T. F-28 yrs.		S.M. M-42 yrs.		S.L. M-44 yrs.	
	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment
Maximum Breath Capacity — Lit./Min.	38.14	63.56	29.66	38.14	19.06	25.43	38.14	52.97
Maximum Breath Capacity — Pct. of Pres.	29%	48%	30%	42%	17%	22%	41%	57%
Vent. at rest Lit./Min.	13.56	15.26	11.02	8.48	8.48	8.48	11.02	8.48
Breathing reserve Lit./Min.	24.58	48.30	18.64	29.66	10.58	16.95	27.12	44.50
Breath. reserve Ratio = $\frac{\text{Br. Res.}}{\text{M.B.C.}} \times 100$	64	76	63	79	55	66	71	84
Vital Capacity — Lit.	2.03	2.46	0.98	1.68	1.10	1.49	2.19	3.27
Vital Capacity — Pct. of Pres.	44%	53%	32%	54%	25%	34%	60%	89%
Oxygen consumed per Lit./Vent.	27cc.	35cc.	27cc.	40cc.	44cc.	68cc.	23cc.	30cc.
Air Velocity Ind. = $\frac{\% \text{ pr. M.B.C.}}{\% \text{ pr. V.C.}}$	0.66	0.90	0.94	0.77	0.68	0.61	0.68	0.64

the ventilatory insufficiency in these cases was an obstruction of the airways. The efficacy of ACTH in these cases may best be explained by the release of the bronchial obstruction which accompanies the bronchial asthma. ACTH may reduce the swelling of the bronchial mucosa caused by allergic or chronic inflammatory changes, thus diminishing the ob-

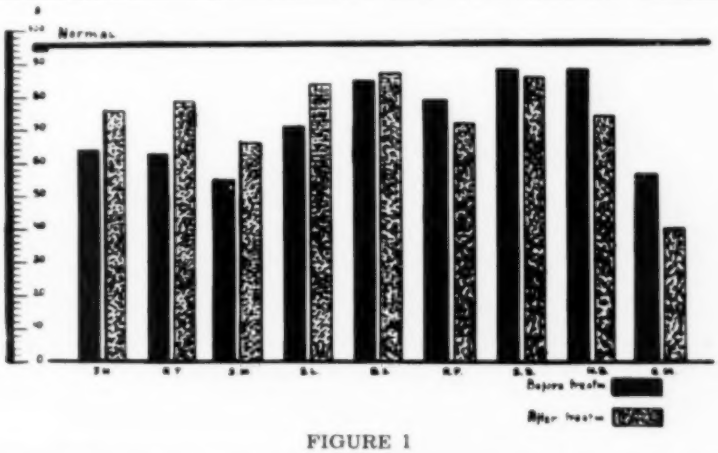

FIGURE 1

TABLE I
THE EFFECT OF INTRAVENOUSLY ADMINISTERED ACTH

B.S. M-42 yrs.		R.F. F-32 yrs.		S.B. F-24 yrs.		N.B. F-44 yrs.		G.M. F-65 yrs.	
Before Treatment	After	Before Treatment	After	Before Treatment	After	Before Treatment	After	Before Treatment	After
88.99	95.34	36.00	33.90	61.44	65.68	72.04	61.49	25.43	21.19
75%	79%	44%	41%	67%	73%	86%	73%	35%	29%
12.71	11.87	7.63	9.32	7.10	8.90	8.05	15.68	11.02	12.70
76.28	83.47	28.37	24.58	54.34	56.78	63.99	45.76	14.41	8.48
85	87	79	72	88	86	88	74	56	40
2.83	3.08	2.02	1.63	1.68	1.95	2.22	2.10	0.75	0.95
61%	67%	70%	57%	56%	65%	67%	64%	21%	27%
34cc.	37cc.	31cc.	36cc.	38cc.	26cc.	33cc.	27cc.	26cc.	24cc.
1.20	1.18	0.63	0.72	1.20	1.10	1.20	1.10	1.70	1.07

struction. Another possibility is that ACTH may counteract the formation of histamine and accelerate its breakdown, thus decreasing spasm of the bronchiolar musculature.[14,15] With the release of the obstruction, alveolar function, too, improved markedly, as may be seen from the increase in the oxygen removal per litre of ventilation. Our results in this group are similar to those reported by Lukas.[9]

The results in case six (R.F.) are difficult to explain. In this case there was some decrease in the maximal breathing capacity and vital capacity, but in spite of this deterioration the oxygen removal per litre of ventilation increased. It should be mentioned that this case showed marked clinical improvement.

Case seven (S.B.) showed improvement of maximal breathing capacity and vital capacity which was not paralelled by an increase in the oxygen removal; on the contrary, this value dropped from 38 cc. to 26 cc. per litre of ventilation. In this case there was a history of long standing asthma and the electrocardiogram showed a pattern of right ventricular hypertrophy. This case was probably on the verge of developing cor pulmonale. During the treatment with ACTH the patient gained 3 Kgm. in body weight. Fluid retention was probably responsible for the development of interstitial or intraalveolar edema which, in spite of the release of the obstruction of the air passages, caused interference with the oxygen exchange in the alveoli.

In the last two cases none of the lung functions improved. In both the air velocity index was higher than one, which indicates a relatively greater reduction of vital capacity in comparison with the maximal breathing capacity. Thus, in these cases diminution of stroke volume—and not bronchiolar obstruction—was the predominant factor in their ventilatory insufficiency (this type of insufficiency has been called restrictive). It may be assumed that in these cases loss of elasticity and other structural changes in the lungs and in the thoracic cage were present at the commencement of treatment, and could not be reversed by the treatment. In this type of ventilatory insufficiency deterioration of lung function has been reported after treatment with ACTH. Thus Galdstone and his associates[8] noted a deterioration of lung functions in a case of emphysema due to old age, and Lukas[9] found a diminution of pulmonary function in a case of long standing and far advanced emphysema and fibrosis.

From our own observations it may be concluded with some reserve that cases of very long standing emphysema and fibrosis with a restrictive type of ventilatory insufficiency will not improve markedly by treatment with ACTH. On the other hand, cases with an obstructive type of ventilatory insufficiency may derive great benefit from treatment with ACTH.

Acknowledgment: We are indebted to Prof. J. Kleeberg, of the Department of Medicine 'A', for placing at our disposal some of the cases referred to in this paper.

SUMMARY

Pulmonary functions were examined in nine cases of bronchial asthma and emphysema before and after treatment with intravenously adminis-

tered ACTH. In five there was an improvement in all lung functions. In the other four cases no uniform response was obtained but most of the lung functions showed deterioration.

RESUMEN

Las funciones pulmonares fueron estudiadas antes y despues del tratamiento de asma bronquial y enfisema con ACTH intravenosa. Este estudio fué hecho en nueve casos. En cinco de estos casos hubo una mejoria de todas las funciones pulmonares. En otros cuatro casos no se obtuvo respuesta uniforme pero la mayoria de las funciones pulmonares mostraron deterioro.

RESUME

Les auteurs ont fait une étude fonctionnelle du poumon dans neuf cas d'asthme et d'emphysème avant et après la mise en oeuvre d'un traitement d'A.C.T.H. par voie intra-veineuse. Dans cinq cas, il y eut une amélioration de la fonction pulmonaire. Dans les quatre autres cas, il n'y eut pas de réponse uniforme, mais dans la plupart, l'examen fonctionnel y révéla une altération.

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Complications of Pneumoperitoneum Therapy*

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During recent years pneumoperitoneum has taken an exceedingly important place in the non-surgical treatment of tuberculosis. This trend in collapse therapy has been well exemplified by the experience in the Municipal Sanatorium. The figures for the past five years are as follows:

	1947	*1948*	*1949*	*1950*	*1951*	*Totals*
Pneumoperitoneum Initiated	25	49	88	175	135	472
Patients Admitted with Pneumoperitoneum	1	2	24	16	46	89
Pneumoperitoneum Refills	543	1,725	3,890	9,024	9,568	24,750

The following list includes all of our known complications:

A. *Thoracic Complications.*
1. Mediastinal Emphysema.
2. Pneumothorax.
3. Bronchial Obstruction and Atelectasis.
4. Marked Pulmonary Collapse.

B. *Complications Due to Administration of Air.*
1. Air Embolism.
2. Subcutaneous Emphysema.
3. Febrile Response.
4. Pain.

C. *Abdominal Complications.*
1. Adhesions.
2. Peritoneal Fluid.
3. Intestinal Obstruction.
4. Gastrointestinal Symptoms and Anatomic Changes.
5. Acute Appendicitis.
6. Tuberculous Peritonitis.
7. Non-Tuberculous Peritonitis.
8. Peritoneal Insult or Mechanical Peritonitis.

D. *Complications of Abdominal Wall.*
Hernia.

E. *Vascular Pressure.*
Lower Extremity Edema.

A. *Thoracic Complications:*

1. *Mediastinal Emphysema.*

There have been two cases with mediastinal emphysema. It occurred early—in one patient after the second, and in the other after the fifth refill. The symptoms were quite typical, not too severe and appeared within

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a few hours after treatment in one patient and after 24 hours in the other. Both complained of swelling of the neck and dysphagia. Physical examination revealed subcutaneous emphysema of the neck and the typical crunching, crackling, systolic sounds over the precordium. Both cases were given oxygen for 24 hours and the pneumoperitoneum was deflated in one. Both showed rapid improvement and within 48 hours the chest findings had cleared and the neck emphysema was practically gone.

The chest roentgenogram was typical with air visualized around the heart and in the retrosternal area.

Pneumoperitoneum was continued in both patients a week after the emphysema cleared. It did not return with treatment.

2. *Pneumothorax.*

We have had two patients with this complication. In one a right partial pneumothorax was visualized on a routine chest film 13 months after pneumoperitoneum was first induced. This took place apparently without loss of air from the pneumoperitoneum space. The pneumothorax caused no symptom and disappeared without treatment in a short time and was not seen on a repeat film 23 days later. Pneumoperitoneum refills were continued without trouble.

The pneumothorax in the second patient occurred in an individual who had received pneumoperitoneum for 11 months without complication. The day after the last refill she felt slight discomfort in the right shoulder and pain in the upper anterior chest. She thought the pneumoperitoneum air was "moving" more than usually. The following morning (48 hours after the refill) while washing (no exertion) she felt tightness and pain in the neck and right chest and shoulder, pressure over the precordium and marked difficulty in breathing. She was in extreme discomfort, and the right chest showed hyperresonance and absent breath sounds. The right chest was deflated without delay—1,200 cc. of air was removed and an indwelling cannula connected to under water drainage was set up. Relief of dyspnoea and chest pain was rapid. After three hours it was possible to discontinue the deflation. That afternoon x-ray film revealed only a residual slight marginal right pneumothorax and a marked decrease and loss of air from the pneumoperitoneum. Apparently the pneumoperitoneum space emptied itself through the right diaphragm into the right pleural space.

Several months before pneumoperitoneum was induced this patient fractured the eighth and ninth right ribs. It is possible that at this time a diaphragmatic injury took place.

We use only the abdominal route left(paraumbilical area) for pneumoperitoneum and not the subdiaphragmatic approach because of the danger of causing pneumothorax by pleural injury with the latter method.

The cases of pneumothorax complicating pneumoperitoneum reported in the literature have all been right sided. It is believed that air from the abdomen can pass through a congenital, inflammatory, degenerative or traumatic defect or rupture of the diaphragm, or it comes through the

diaphragmatic openings to reach the mediastinum and then breaks through the interstitial tissues into the pleural space.

3. *Bronchial Obstruction and Atelectasis.*

(a) Large bronchus obstruction: Pneumoperitoneum can have a direct influence on the bronchial lumen and bronchial drainage. With the elevation of the diaphragm by pneumoperitoneum, the rotation, or direction of the bronchi can be altered. If the bronchus is the site of a severe edematous or infiltrative process, complete obstruction of the airway may result. Symptoms and findings typical of bronchial obstruction occur with severe dry cough, wheezing, occasional dyspnea and bouts of fever and constitutional symptoms. In view of the marked impairment of bronchial drainage, continuation of pneumoperitoneum in these patients is contraindicated.

We have had three cases with obstruction of a major bronchus. In all of them an intrinsic endobronchial tuberculous lesion was present. In each instance phrenic crush had been done on the side where the obstruction developed. The pneumoperitoneum, supplemented by the phrenic crush, aggravated and completed the bronchial narrowing and obstruction. Streptomycin and para-aminosalicylic acid may not be able to eliminate the obstruction as long as the pneumoperitoneum is continued.

(b) Small bronchus obstruction: We have had nine instances of segmental atelectasis. In all of them atelectasia was in the form of a heavy linear segmental band immediately above the diaphragm. Eight of these nine patients had had phrenic nerve crush to supplement the pneumoperitoneum and in all atelectasia appeared after the operation. In only one was there symptoms of possible bronchial obstruction with cough, wheezing and fever. In this case bronchoscopy revealed redness and exudate, and these findings later cleared with antibiotics.

These linear atelectatic areas usually cleared when the pneumoperitoneum was relaxed for a time by giving fewer refills or less air.

Our experience in these cases indicates that the phrenic nerve should be crushed only for special indications and after its potential hazards have been considered. In many patients after phreniclasia, complete or partial diaphragmatic paralysis remains. Moreover, atelectasis or bronchial obstruction occurs most often in those with phrenic operations. Phreniclasia should be done if the lesion requires the additional lung collapse to become inactive and if it is intended to use the pneumoperitoneum as the definitive form of treatment. Phrenic crush is not necessary if the lesion is being controlled by pneumoperitoneum alone.

Alleviation of endobronchial tuberculous lesions with antibiotics before instituting pneumoperitoneum should also be considered.

4. *Marked Pulmonary Collapse.*

It is possible to get a considerable degree of pulmonary collapse with pneumoperitoneum. True, with added phrenic nerve paralysis a greater elevation of the diaphragm can be obtained. With the diaphragms elevated pulmonary function can be diminished and with bronchial narrowing, symptoms like dyspnea or wheezing can occur (abdominal pain and other

gastrointestinal symptoms can also be present). It must be emphasized that the best pneumoperitoneum is the one with the smallest degree of pulmonary collapse that controls the diseased areas. We have had an occasional case where the pneumoperitoneum caused pressure phenomena, and relaxation of the collapse relieved symptoms.

B. *Complications Due to Administration of Air.*

1. *Air Embolism.*

During the past five years almost 25,000 refills have been given to our patients and 472 pneumoperitoneums initiated. Fortunately in this period we have had only two instances of air embolism or peritoneal shock with recovery.

The air embolism occurred in a well established pneumoperitoneum space during the patient's 11th refill. After 750 cc. of air had been given the patient suddenly developed a generalized convulsion and became unconscious. The pulse was imperceptible and the dilated pupils did not react to light. Treatment consisted of Trendelenburg position, oxygen, caffein and small repeated doses of adrenalin. In about 15 to 20 minutes the patient became conscious, and within an hour had apparently recovered completely. After a week air was again given and the pneumoperitoneum continued a month until the patient was transferred for surgery. The pneumoperitoneum reexpanded completely during this period. Five months after the original episode of air embolism, pneumoperitoneum was reinduced without difficulty. During the second refill (100 cc. given) a reaction occurred with slight convulsive seizure, dyspnea and sweating. With coramine and oxygen the patient recovered rapidly. Pneumoperitoneum was discontinued.

All of our treatments are given in the left paraumbilical area (to avoid the liver). However, the needle can enter an adhesion, the omentum or spleen. The patient should always be in Trendelenburg position whenever air is given. Care must be used to be sure a free airway is present with no blood in the needle. A recent recommendation in treatment of air embolism is to place the patient in the left lateral position to avoid an acute obstruction in the form of an air trap in the right ventricle and interference with outflow of blood.

2. *Subcutaneous Emphysema.*

This was only observed a few times, did not last long and was of no clinical importance.

3. *Febrile Response.*

We have had two cases with febrile response noted immediately after induction of pneumoperitoneum. (There are many more patients with fever and symptoms occurring soon after pneumoperitoneum is initiated. These are described below in discussion of peritoneal complications.) In both patients the temperature was high during the first four days and then fell by lysis and became normal in 10 days. In both cases the air was visible only under the left diaphragm during the first 48 hours and then

appeared under the right diaphragm as the liver began to separate from it. One complained of pain in the right shoulder and over the right lower ribs when this happened—"felt the air move over." The other had nausea, headache and diffuse tenderness over the abdomen, especially the right side. Symptoms gradually cleared in both patients and pneumoperitoneum was continued in one case for seven months when sanatorium discharge took place, and in the other for only one month when it was stopped because of recurrent abdominal pain and ineffective collapse. The episodes may have been related to the separation of the liver from the diaphragm with the tearing of adhesions or irritation of the site of an old peritoneal infection.

4. *Pain.*

Pain experienced with pneumoperitoneum is of several types. (a) Immediately after induction most patients have shoulder pain. This usually disappears after a few days. (b) Quite a number have episodes of generalized abdominal discomfort or pain, usually associated with pressure of the pneumoperitoneum. Pain can also be due to peritoneal complaints. (c) In this discussion we refer only to pain of such severity as to cause temporary or permanent discontinuation of the treatment. We have had four such cases. The severe pain occurred within one week, several weeks, three months, and one year respectively, after the induction of pneumoperitoneum. The pain was epigastric and paraumbilical in location. Refills were stopped in three patients for three, four and seven weeks respectively, and then resumed. This was without difficulty in two but severe pain recurred in the third and pneumoperitoneum was discontinued. In the fourth, with a history of duodenal ulcer, refills were abandoned because of extreme abdominal distress and pain.

C. *Abdominal Complications.*

1. *Adhesions.*

Adhesions are present in the majority of patients. In a series of 100 consecutive cases, they were observed in 70 per cent. They can be in the form of strings or bands, or organs like the liver and spleen are adherent. We found about equal numbers of string, band and combination of string and band adhesions. The liver and spleen were adherent in 4 per cent. Most of the adhesions are thin. In only about 12 per cent are broad, wide or apron adhesions found. The great majority of adhesions are not long enough to reach the diaphragm domes and most of them appear much lower. We found that the string and band type in all but 2 per cent of cases had no influence on elevation of the diaphragms and did not interfere with its rise. On the other hand, in cases with adherent liver and spleen, an effect on the diaphragms was usually evident and the diaphragmatic rise was definitely limited. When the liver and spleen are adherent there are usually numerous broad bands about both organs which bind them down.

There may be symptoms associated with adherent organs such as pain,

anorexia, or abdominal distress. String adhesions can stretch, permitting the abdominal organs to move downwards. The liver and spleen can be gradually pressed away from the diaphragms which can show some degree of elevation. On occasion, with the liver and spleen adherent, a good pneumoperitoneum space is obtained by the downward depression of the remaining organs. In patients with adherent liver and spleen we have often elicited a history of a preceding abdominal, or less often, pelvic operation or previous infection, such as peritonitis or pelvic inflammatory disease. Adherent organs have also followed peritoneal effusions and in one case appeared after pleurisy with probable diaphragmatic inflammation. Sometimes no history of operation or infection is obtained, yet adherent organs are present.

2. *Peritoneal Fluid.*

Fluid in the pneumoperitoneum space varies in amount and type. Usually only little fluid is found and it is unassociated with symptoms. Less often the fluid is of considerable degree and causes pressure phenomena, such as fullness or pain in the abdomen or back, and dyspnea. Even more rarely, in addition to pressure symptoms, the patient is acutely ill and toxic with malaise, fever, sweats, nausea, vomiting and diarrhea. In such cases, tubercle bacilli may be found in the fluid.

Peritoneal fluid has been treated according to the following methods: (a) Usual refills; (b) less air; (c) fewer refills; (d) less air and fewer refills; (e) temporary discontinuation; (f) deflation; (g) aspiration of abdominal fluid; (h) removal of fluid and administration of antihistaminics; (i) permanent discontinuation.

We have had 27 patients with fluid in the pneumoperitoneum space who have been observed and treated with the following results:

	Patients
(1) Eighteen with a small to moderate amount of fluid and no symptoms (most patients with small effusions) treated by	
(a) Usual refills and fluid resorbed	9
(b) Less air and fewer refills with resorption of fluid	7
(c) Discontinuation of pneumoperitoneum for a few weeks and resorbed	1
(d) Less air but fluid remained (until patient's discharge)	1
(2) Three with moderate amount of fluid and slight pressure symptoms:	
(a) Treated with a few aspirations and less air and fluid stopped reforming	1
(b) Fluid aspirated and refills stopped for one month. Resumed and no more fluid formed	1
(c) Pneumoperitoneum stopped (early in series)	1
(3) Three with tubercle bacilli in fluid:	
(a) Patients acutely ill with much fluid and pressure phenomena. Treated with deflation of air and aspiration of fluid (antihistaminics to one patient in addition). Fluid reformed in spite of repeated taps and condition controlled by discontinuation of refills	2
(b) One was admitted with abdominal fluid, positive for tuberculosis (culture). Although asymptomatic, the pneumoperitoneum was discontinued	1

(4) Three with much fluid and pressure symptoms treated with less air and frequent aspirations of fluid but the fluid kept reforming. Then tapped dry and given antihistaminics because a high percentage of eosinophiles were found in the fluid and an allergic etiology considered:
(a) Very little fluid reformed 2
(b) Fluid did reform 1

From these figures it is evident that pneumoperitoneum effusions are usually slight in degree and do not cause symptoms. The majority of these effusions clear with refills continued in the usual manner or with decrease in amount of air and frequency of refills. When considerable fluid forms with pressure symptoms, aspirations are necessary and should be repeated until the abdomen is free of effusions. The refills can then be decreased in amount or stopped for a while and later continued in the usual fashion. If fluid keeps reforming in spite of aspirations then the abdomen should be emptied and an antihistaminic given while the pneumoperitoneum is continued. If the patient is acutely ill it usually means the presence of tuberculous peritonitis. The fluid should be aspirated and the pneumoperitoneum stopped.

In nine of the above patients the abdominal fluid was aspirated and examined. In all it had a high specific gravity, many cells and a total protein content of about 5 grams per cent. Seven of the nine patients had a high percentage of eosinophiles in the fluid. Four of them have been the subject of a separate report[1] dealing with the use of antihistaminics in pneumoperitoneum effusions. In them it was found that repeated paracenteses failed to control the fluid as it reformed rapidly. Then removal of the fluid was combined with the use of an antihistaminic and in two, little fluid reformed. The value of the antihistaminic was tested by discontinuing the drug and in each instance the fluid reformed rapidly. When it was removed a second time and the antihistaminic reinstituted only a small amount of fluid appeared. The antihistaminic does not influence the absorption of fluid and when given alone the fluid will not clear. However, if it is first removed, the drug apparently keeps it from reforming.

3. *Intestinal Obstruction.*

We have had two patients with intestinal obstruction. The first had an 18 year history of peptic ulcer. Fourteen months after the initiation of pneumoperitoneum he had an acute episode with symptoms typical of intestinal obstruction. With deflation of the pneumoperitoneum, Wangensteen drainage of stomach, intravenous fluids and rectal tube, the condition cleared after 36 to 48 hours. However, refills were discontinued. X-ray examination 10 days after this episode revealed an acute partially obstructing duodenal cap. Two months later the obstruction was not visualized.

The second case had an acute abdominal attack 14 months after pneumoperitoneum was initiated. This consisted of severe cramps all over the abdomen, retching, vomiting and diarrhea. The temperature was normal and there was no leucocytosis. Examination revealed marked distention

and tenderness of the abdomen. Treatment consisted of deflation, intravenous fluids, Wangensteen suction of stomach and rectal tube. After two days the condition was relieved. A similar attack occurred a week later which subsided after one to two days' treatment. Once more after a week's interval, a third attack occurred which left some distention, slight discomfort and occasional vomiting. The patient was transferred to Bellevue Hospital where exploratory laparotomy was done. The operative findings have been kindly furnished by Dr. Donald A. Davis:

"At operation the peritoneum was found to be covered by a pseudoperitoneum or membrane, about one-eighth of an inch thick, which covered all surfaces of the intestinal and parietal walls and encased the entire small bowel in an envelope. There was no free fluid in the abdomen. The pseudoperitoneum which contained the distal ileum was excised and a Nobel procedure was done.

Since the operation the patient has had no recurrence of obstruction.

"The pathological report on specimen of the pseudoperitoneum follows: Sections reveal a mass of dense hyalinized fibrous tissue with many fibroblasts. There is no tuberculosis. Diagnosis: Fibrous tissue with chronic inflammatory reaction."

The intestinal obstruction was obviously caused by the fibrinous membrane. Apparently the pneumoperitoneum, by its irritation, caused the laying down of this fibrous tissue. There is additional evidence mentioned below in discussion of other peritoneal complications that pneumoperitoneum can cause formation of fresh non-inflammatory exudate. In either case it is plausible to believe that adhesions and intestinal narrowing can result from such material.

4. *Gastrointestinal Symptoms and Anatomic Changes.*

The most common disturbance associated with pneumoperitoneum is loss of weight. We have noted this in about 60 per cent of our patients. In a series of 100 consecutive cases who remained in the sanatorium at least one year after pneumoperitoneum was induced, the weight changes were checked every three months with the following results:

	Gain in Weight	Loss in Weight	No Change in Weight
3 Months	34	56	10
6 Months	34	60	6
9 Months	34	64	2
12 Months	30	62	8

Loss or gain in weight is often progressive (this was noted in 40 per cent of our cases), until weight stability is achieved. In the majority of cases the change in weight was usually below 10 pounds. Although a few gained more than 10 pounds, a considerable number lost more than 10 pounds and up to 25 pounds. To overcome this loss of weight, it would be advisable to give small regular meals and feedings between meals.

Most patients do not have important gastrointestinal symptoms. True, many with weight loss have some anorexia. Yet loss of weight may occur with no obvious complaint. Some may have mild symptoms (eructations,

diarrhea, abdominal pain, belching and nausea) for a day or two at a time, but it is uncommon for them to have persistent, severe complaints.

We have found the following x-ray changes: (a) Elongation and narrowing of the cardiac end of the stomach. (b) Widening and dilatation (atonicity) of the fundal portion of the stomach. (c) Drop of the stomach. (d) Frequent anterior displacement of the stomach with occasional anterior rotation. (e) Increase or decrease in intestinal motility.

5. *Acute Appendicitis*

We have had two cases of acute (gangrenous) appendicitis, occurring two and 15 months respectively, after pneumoperitoneum was induced—one with appendectomy and recovery, the other with appendiceal rupture, peritonitis and death. This incidence is not greater than that found in our patients without pneumoperitoneum. However, acute appendicitis can be a dangerous complication and there are several important clinical points to be emphasized in relation to this problem. The symptoms can vary much and be atypical and the physical findings can be uncharacteristic. Fever appeared early in both of our cases, with nausea in one, and no nausea or vomiting in the other. One had diarrhea for a few days, the other not. After 48 hours nausea, retching and vomiting was present in both patients. Slight distress of the lower abdomen was evident but no localized pain in the right lower quadrant. The patients appeared acutely ill with the abdomen distended. There was no localized tenderness in the right lower quadrant (deep or rebound) and no rigidity of the abdominal muscles. However, rectal or pelvic examination revealed a definite mass in the appendiceal area which was tender. The leucocyte count was elevated in one case and not in the other but in both there was a marked elevation of the non-segmented cells.

These cases brought out the following points: Abdominal examination can be misleading in the diagnosis of acute appendicitis. The air apparently prevents the usual right lower quadrant pain, tenderness, and rigidity, and interferes with localization of the infection. To facilitate the physical examination and to get a more accurate appraisal of the possibility of appendicitis, the abdomen should be deflated as much as possible without delay. The rectal or pelvic examination provides the most important finding—fullness or tenderness in the appendiceal area. The blood count is significant, as there is marked elevation of non-segmented cells. In any patient with fever, abdominal distention or atypical symptoms, acute appendicitis should be carefully considered. If there is any doubt about the diagnosis, there should be no delay in doing exploratory laparotomy.

6. *Tuberculous Peritonitis.*

We have had two cases of tuberculous peritonitis with effusion. These were mentioned above under pneumoperitoneum effusions. It is not proper to draw conclusions from two cases, yet these had characteristics which made them distinctive compared to the others with effusions. In both cases an acute clinical course was associated with the fluid and peritonitis. The onset was two and one-half and eight and one-half months after the

initiation of pneumoperitoneum respectively. The symptoms consisted of fever, nausea, vomiting, pain in abdomen and back, and diarrhea. They appeared toxic and their abdomens were tender. The fluid had the characteristics of an exudate and was positive for tubercle bacilli on culture and guinea pig inoculation. Antibiotics (streptomycin and para-aminosalicylic acid) influenced favorably the temperature and toxicity. However, deflations of air and aspirations of fluid did not definitely control the condition though the patients were made more comfortable. The fluid continued to reform until the pneumoperitoneum was discontinued. In both cases the fluid became pocketed. Our experience would indicate that in the presence of tuberculous effusion, the wisest policy is to remove fluid (and air) and discontinue refills.

7. *Non-Tuberculous Peritonitis.*

We have had one patient with non-tuberculous peritonitis and effusion who was transferred to Otisville 16 months after initiation of pneumoperitoneum. She was admitted in an acutely ill and toxic condition. The abdomen was distended and showed a fluid wave. There was (after deflation) slight, poorly defined abdominal tenderness. Diagnostic aspiration revealed abdominal pus (cloudy, greenish and sickening sweet odor) with a profuse growth of staphylococcus albus and a scant growth of coliforms. Tubercle bacilli were not found. Laparotomy was done two weeks after admission. The surgeon noted "Peritoneum thick and edematous. Four thousand cc. creamy white pus suctioned out and one to two pounds cheesy, wet, creamy necrotic tissue. Entire pus and necrotic tissue contents lay in a pocket formed by anterior abdominal wall anteriorly, omentum posteriorly, liver superiorly and fusion of omentum to pelvic peritoneum inferiorly." The temperature came down by lysis after the operation and the patient recovered. Pneumoperitoneum was discontinued. The exact etiology of this peritonitis was not determined.

8. *Peritoneal Insult (Mechanical Peritonitis).*

This term is used for want of a better one and includes cases with peritoneal irritation but indefinite diagnoses. We have had five such patients. These acute peritoneal episodes occurred early after induction of pneumoperitoneum two, four, 15 and 17 days, and two months respectively. Symptoms included fever in four, abdominal pain in three, nausea in three and vomiting in one. Physical examination revealed abdominal tenderness in five (especially right lower quadrant in three), lower abdomen rebound tenderness in three and spasticity in right lower in two. Rectal examination was negative except for slight tenderness and fullness in one. Blood count showed slight leucocytosis in three and definite increase in one. An increase in non-segmented cells (up to 25) was found in two cases.

In one with supportive treatment and deflation and then discontinuation of pneumoperitoneum, the condition cleared rapidly. In the other four cases the possibility of appendicitis was considered and they were sent to a local general hospital for surgical observation or treatment. In three of them the condition subsided in a few days and no operation was per-

formed. The fourth had appendectomy. One of these patients had abdominal fluid at the time of this peritoneal involvement which may have been related to the symptoms. Pneumoperitoneum was not continued in this case.

Pneumoperitoneum was also discontinued in the other two patients after the peritoneal episode. However, in both of them it was reinduced at a later date. In one this was done 20 months after the acute illness. A good pneumoperitoneum space was obtained. After three months a small amount of fluid appeared. The patient was comfortable except for one episode of abdominal and back pain. Pneumoperitoneum was continued in the usual manner without trouble.

In the other patient pneumoperitoneum was reinduced after eight months. The day it was started abdominal pain was experienced. In spite of only small amounts of air, pain persisted. Some tenderness and spasticity was found in the upper abdomen. The temperature rose to 100 degrees F. for one day. Symptoms persisted for eight days. After 10 days all complaints and physical findings had cleared. It was possible then to give the usual refills. A good space was obtained and refills continued with no further difficulty.

In the case with appendectomy, the surgeon reported "no free fluid in the abdomen. The appendix was not unusual. The intestines and mesentery were covered with thin greyish exudate. Microscopic tissue showed congestion of the serosal capillaries." There was just hyperemia present and no evidence of recent inflammatory change. The appendix also showed only congestion of the serosal capillaries.

This case illustrates that pneumoperitoneum can be irritating and cause deposition of exudate, i.e., a mechanical peritonitis. Those in this group all had peritoneal symptoms soon after therapy was started. In the operative case the exudate was thin and without inflammatory change. Because of the peritoneal irritation, pneumoperitoneum can cause symptoms of various kinds. All of the patients in this group had evidence of acute peritoneal insult. On a long term basis, with sufficient exudate and fibrinous material present, other symptoms can occur, adhesions can form or even intestinal obstruction can result. On the other hand, the peritoneal involvement may subside so that after waiting a sufficient length of time, pneumoperitoneum can be reinduced and continued effectively without difficulty.

D. *Complications of Abdominal Wall: Hernia.*

We have had 16 cases of herniation associated with pneumoperitoneum which can be classified in three groups.

Group 1: Patients with repair of herniation before pneumoperitoneum therapy. There were four males, with one direct and three indirect inguinal hernias. After herniorrhaphy, pneumoperitoneum was instituted at intervals of two and one-half, three, five and seven months respectively. In the beginning of our pneumoperitoneum work our surgeon was conservative concerning the delay in starting treatment after operation. A wait of four

weeks would usually now be considered adequate. It would be advisable to give the treatments cautiously for a while. The refills were continued without difficulty in our four patients for eight, 11, 12 and 18 months respectively.

Group 2: Patients with pneumoperitoneum therapy in presence of old herniation. There were two men in this group. One with old bilateral, indirect inguinal herniation for many years. Pneumoperitoneum was induced and continued for 27 months without difficulty. He has been wearing an abdominal belt (up to four months ago had used a truss). He refused herniorrhaphy and is confortable with pneumoperitoneum. The other had right direct inguinal hernia for years. After 10 months' pneumoperitoneum pain appeared over the hernia. It appeared slightly larger and herniorrhaphy was done. Pneumoperitoneum was reinduced two and one-half months after operation and has now been continued seven months without difficulty.

Group 3: Patients with herniation developing during pneumoperitoneum. This group consisted of 10 patients. Included were three umbilical, three pneumatoceles of the spermatic cord and four inguinal hernias. Two of the three umbilical cases were in females and all the remaining patients were males.

The umbilical hernias appeared at two to four months after pneumoperitoneum was started. Two had herniorrhaphy and one did not. The hernia was present one year before operation in both cases. In the patient without operation, the pneumoperitoneum was continued 14 months without difficulty and with no symptom. In one the hernia became painful with slight increase in size and some discoloration of the skin. After the repair pneumoperitoneum was discontinued because of fluid in the abdomen. In the other (information kindly furnished by Dr. C. Lester) the hernia had been present a long time and was reducible, but during an attack of grippe, with severe paroxysmal cough, the umbilical hernia became incarcerated. The swelling had become larger, was tender and nonreducible and the overlying skin was discolored. After the repair, pneumoperitoneum was continued. This is the only one with post-sanatorium observation and an operation after discharge but was included because of unusual interest.

The three pneumatoceles of the spermatic cord appeared at intervals of four, 20 and 21 months respectively, after pneumoperitoneum was started. Two had no herniorrhaphy and pneumoperitoneum has been continued for two and six months respectively, since the appearance of the hernia. One is asymptomatic and the other complains occasionally of slight pain. In the third, repair was done two months after the hernia became evident. The herniorrhaphy was recommended because the hernia had become tense and painful though it was reducible. Pneumoperitoneum was reinduced one month after operation. The refills were given regularly each week in the routine manner except that they averaged only 800 cc. each during the first two months instead of the usual 1,000 cc.

Of the inguinal hernias, three were unilateral and one bilateral. They

appeared at intervals of six, seven, eight and 26 months respectively after initiation of pneumoperitoneum. Two had no repair and two did, one and five months respectively, after the appearance of herniation. The two without operation had pneumoperitoneum continued for two months after herniation appeared when they left the institution without symptoms. In the two operative patients pneumoperitoneum was stopped after the herniorrhaphy in one, while in the other (bilateral hernia) it was reinduced four months after operation. One of these patients had no hernial symptoms and one had slight occasional pain which was increased by cough and straining.

Our experience in these groups would indicate that herniation is not necessarily a contraindication to the use of pneumoperitoneum. In many clinics patients are examined routinely for herniation before induction of collapse. The hernia can often be controlled during pneumoperitoneum therapy by the use of a truss or belt. It would be advisable, however, to repair the hernia whenever possible. Pneumoperitoneum can then be induced in a short time without difficulty. The occurrence of herniation during pneumoperitoneum therapy does not mean that treatment must be stopped. It is possible to continue therapy with or without herniorrhaphy.

E. *Vascular Pressure: Lower Extremity Edema.*

There have been three females and one male with edema of lower extremities. In the three women edema appeared early after induction of pneumoperitoneum, within one month in two, and after two months in the third. Pneumoperitoneum had been carried on in the usual fashion in these cases. Edema varied in each patient and extended up to the ankles in one, involved the feet and legs in another, and in the third, the swelling was marked, involving the entire lower extremities. There was also frequency and urgency and occasional pain in the groins. Gynecological, cardiological and urological examinations in all of these patients were negative. In the mildest case edema disappeared rapidly with little change in the refills. In the other two, refills were stopped for 10 to 14 days and then resumed in small amounts for several weeks. Under this regime edema in both patients gradually disappeared in two weeks and did not return.

The only man in this group had been given much air and his abdomen was distended. Edema of the feet and ankles occurred eight months after initiation of pneumoperitoneum. Urological and cardiological examinations were negative. The abdomen was deflated and the edema cleared within five days. The refills were diminished in amount and given less often. This was continued for two months and then usual refills were resumed. Edema did not return.

With pneumoperitoneum, the viscera are crowded into the pelvis. The probable explanation for edema is the pressure exerted on the large pelvic veins. The lack of pelvic, cardiac or nephritic pathology in these patients and the clearance of the condition by decompression of the pneumoperitoneum would tend to bear this out.

SUMMARY

We have outlined the complications which have occurred in a large sanatorium group of patients treated with pneumoperitoneum. Our observations have been limited to the institutional phase of treatment. The number and incidence of complications would probably be greater if post-sanatorium follow-up had been included. There is no question about the value of pneumoperitoneum in the treatment of pulmonary tuberculosis. It has a popularity that is well deserved. However, this study has shown that there are many associated complications. Some of them are mild but others are serious. Pneumoperitoneum is, therefore, a procedure that should not be recommended lightly as treatment that is without hazard. Before it is induced the indications should be carefully considered and the reason for its use should be important.

RESUMEN

Hemos señalado las complicaciones que han ocurrido en un gran sanatorio en un grupo de enfermos tratados con neumoperitoneo. Nuestras observaciones se han limitado a la fase institucional del tratamiento. El número y la frecuencia de las complicaciones sería probablemente mayor si el seguimiento de los enfermos después de abandonar el sanatorio se hubiese incluido. No hay duda respecto del valor del neumoperitoneo en el tratamiento de la tuberculosis pulmonar. Tiene una popularidad que bien merece. Sin embargo, este estudio ha mostrado que tiene muchas complicaciones asociadas. Algunas de ellas son benignas pero otras son serias. El neumoperitoneo es por tanto un procedimiento que no debe recomendarse a la ligera como tratamiento sin peligros. Antes de iniciarse las indicaciones deben ser cuidadosamente consideradas y la razón para usarlo debe ser importante.

RESUME

Nous avons esquissé les complications survenues dans un groupe étendu de malades traités par pneumopéritoine en sanatorium. Nos observations ont été limitées à la période d'institution du traitement. Le nombre et l'incidence des complications seraient probablement plus élevés si l'on avait pu suivre complètement les malades après leur sortie de sanatorium. La valeur du pneumopéritoine n'est pas en question dans le traitement de la tuberculose pulmonaire. Il à une popularité qu'il mérite bien. Cependant cette étude a montré qu'il y a beaucoup de complications associées. Quelques-unes d'entre elles sont bénignes mais d'autres sont sérieuses. Le pneumopéritoine est, par conséquent, un procédé qui ne devrait pas être conseillé à la légère, comme un traitement qui ne peut être grevé d'aucun incident. Avant qu'il ne soit entrepris, ses indications devraient être considérées avec soin, et les motifs qui déterminent son emploi solidement basés.

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Mucocele of the Lung Due to Congenital Obstruction of a Segmental Bronchus: A Case Report; Relationship to Congenital Cystic Disease of the Lung and to Congenital Bronchiectasis*

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The case of mucocele of the lung herein reported is of interest primarily because of its probable relationship to bronchogenic cyst on the one hand, and congenital bronchiectasis on the other. The author suspects that it is, in fact, the same condition as the former, but in an early phase, and that either is antecedent to true congenital bronchiectasis.

Case Report

Miss M.S., Orange County General Hospital. This 15 year old Mexican female was admitted to the hospital March 21, 1949, with symptoms of weakness, easy fatigue, and feeling feverish. There was weight loss of 10 pounds over the previous two months and history of close contact with active tuberculosis in the preceding two years. Physical examination was not remarkable; there were slightly increased breath sounds at the right apex. X-ray films of the chest showed bilateral infiltrations of moderate degree and a circumscribed lesion in the left lung. She reacted to tuberculin. The sputum was negative, but a gastric culture was positive, establishing definitely the diagnosis of pulmonary tuberculosis.

On a regimen of bed rest in the hospital, the pulmonary infiltrations decreased markedly, with the exception of the rounded density in the region of the apex of the lower lobe on the left side. X-ray films taken at several distinct intervals show this to persist (Figures 1 and 2). It was featured by an air-fluid level.

Because of this, the patient was seen in consultation, and it was considered that the rounded lesion should be removed surgically, the tentative diagnosis being either a congenital bronchial cyst with associated tuberculosis, or pulmonary tuberculosis with cavitation in the left lower lobe. On July 22, 1950 findings at operation were as follows:

There were filmy adhesions between the superior and posterior segments and the adjacent chest wall. The arrangement of the lung was abnormal in the following respects: the basal segments were divided into two equal portions by a long vertical fissure; there was a partial fissure between the superior segment of the lower lobe and the basal segments; the interlobar fissure was incomplete, being totally absent in the posterior one-half; the bronchi of the posterior segment of the upper lobe originated from a subsegmental bronchus (B6b)[1] of the superior segment of the lower lobe, as illustrated, and thus traversed the posterior portion of the usual fissure line.

Dissection revealed a smooth, rounded, pale, semitranslucent cyst-like dilatation of the bronchial system, extending through the superior and posterior segments. When an occasional branch was ruptured during the dissection, it was found to contain a large amount of thick, whitish, mucoid material (Figure 3). There was absolutely no evidence of inflammation or of solid deposit.

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Presented at the scientific meeting of the Department of Surgery, U.C.L.A. School of Medicine, February 12, 1952.

After division of the superior segmental bronchus of the lower lobe just distal to the origin, it was possible to visualize the proximal stump lumen. This was imperforate, resembling a shallow, blind, funnel-shaped pocket, the walls of which were completely smooth as were the walls of all the cystic spaces mentioned above. The entire lung had relatively little carbon deposit, but the superior segment was totally devoid of such pigmentation. All segments of the lung seemed to inflate normally, although the posterior segment deflated somewhat more slowly than the other. The blood supply, artery and veins, and the remaining hilar and pulmonary anatomy were normal. The diaphragm, pericardium, and mediastinal structures also were normal.

There was a 2 cm. focus of induration palpable beneath the surface of the superior lingula segment. On removal this was found to be somewhat similar to the above abnormalities, but the bronchi to this subsegment were not dilated and contained no mucoid material. No obstructing septum was present. The bronchial walls here were thickened, more fibrotic and somewhat nodular, and in one place there was a white amorphous deposit, approximately 1 mm. in diameter. Pathologically this proved to be tuberculosis.

The operation performed was a triple pulmonary segmental resection of the left lung, removing the superior segment of the lower lobe and the posterior segment of the upper lobe together, and a subsegment of the superior lingular segment of the upper lobe separately, because of the tuberculous lesion at that point. Her convalescence was uncomplicated. She is still in the Sanatorium with active tuberculosis.

On November 18, 1950 she was bronchoscoped in an attempt to visualize the superior segmental orifice of the left lower lobe. Such a superior segmental orifice was not visible. The remaining tracheo-bronchial tree was within normal limits. The features of the case are:

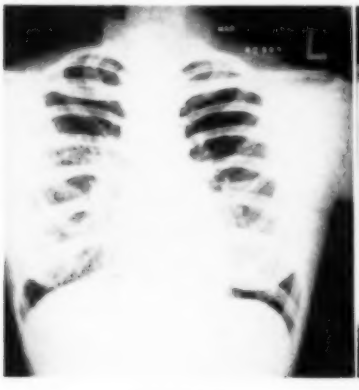

FIGURE 1

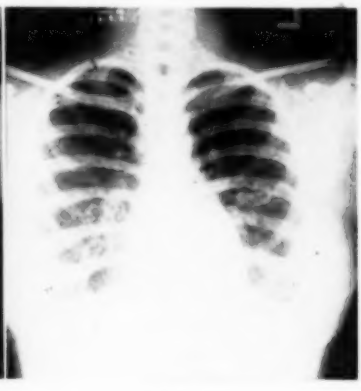

FIGURE 2

Figure 1: Radiograph dated March 22, 1949. In the third left anterior interspace, there is a round lesion containing fluid in the lower one-half and air in the upper half. Note that the wall is extremely thin. There is also associated bilateral pulmonary infiltration. — *Figure 2:* Radiograph taken July 1, 1950. This shows the same round lesion in the third left anterior interspace. The wall is still extremely thin. The relative amounts of air and fluid have changed slightly. The size of the lesion has not changed. The bilateral infiltrations have decreased considerably.

1) The diaphragm-like obstruction at the commencement of the superior segmental bronchial orifice.

2) The distention, with glairy mucus, of the entire bronchial system tributary to this (all three branches of the superior segment of the lower lobe and also the posterior segment of the upper lobe) (Figure 4).

3) The distention with air of the alveoli of the tributary segments, in spite of the filling with mucus of the bronchial air ducts and the complete obstruction of the inlet orifice.

4) The normal size, and inflation-deflation of the alveoli of the tributary segments.

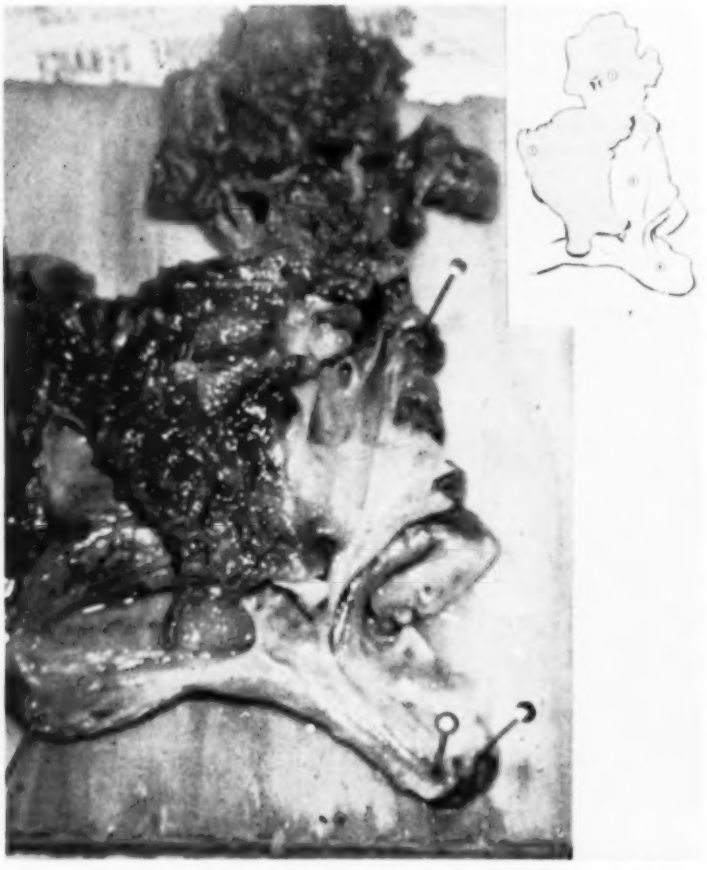

FIGURE 3: Photograph of gross specimen, showing the bronchus of the posterior segment of the upper lobe leading into the bronchus of the superior segment of the lower lobe. The insert explains the photograph: (1) the posterior segment of the upper lobe; (2) the bronchus of the posterior segment of the upper lobe; (3) part of the superior segment of the lower lobe; (4) the main stem bronchus of the superior segment of the lower lobe at the site of amputation.

5) The lack of densities conforming to the pathological findings on x-ray examination, one air-fluid cyst alone being demonstrated.

6) The air component of the air-fluid cyst in spite of a totally blocked bronchial system.

7) The lack of pigmentation in the involved segments.

8) The aberrant drainage of the segmental bronchus from the posterior segment of the left upper lobe into the subsegment bronchus (B6b) of the superior segment of the lower lobe.

9) The use of segmental resection allowed removal of the pathological areas with loss of only two segments, whereas lobectomy surgery would have meant the sacrifice of the entire lung.

Discussion

Sante,[2] following Mueller's speculation, has well described the probable method of formation of congenital bronchial atresia, partial or complete. The sequence of events is considered to be that a bronchial bud fails during its growth to have normal tubular development. If the failure is complete, the bronchiole becomes represented only by a cord. If growth is resumed after a time, tubularity is established distal to an area of artesia. If the bronchiole so obstructed develops secretory glands, the fluid so formed will accumulate with the production of a fluid cyst.

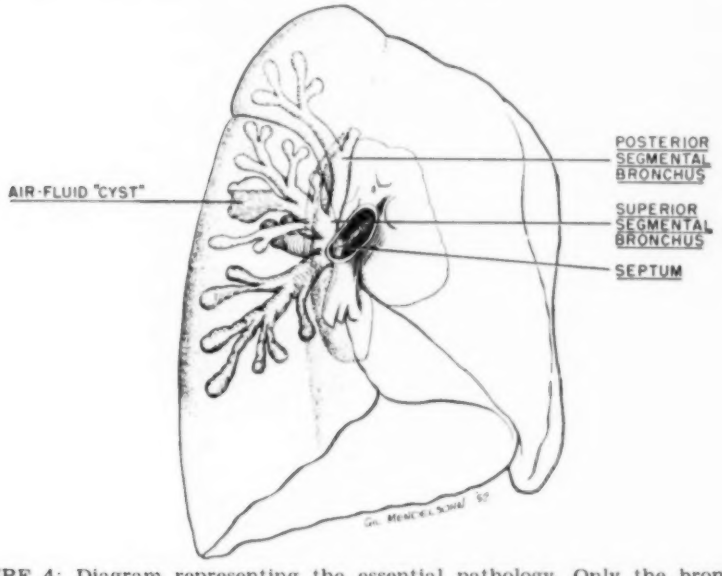

FIGURE 4: Diagram representing the essential pathology. Only the bronchial systems of the two affected segments are illustrated. The bronchi of the posterior segment of the upper lobe and the bronchi of the superior segment of the lower lobe have a common obstruction, the septum at the commencement of the superior segmental orifice of the lower lobe. All the bronchi tributary to the obstructing septum are dilated and filled with mucus. One small branch, however, contains air in addition to mucus. The parenchyma of both the affected segments is in a normal state of inflation, despite the bronchial obstruction.

The thin, smooth diaphragm at the orifice of the superior segmental bronchus was not removed for obvious reasons, but from bronchoscopic study and the appearance at surgery when it was exposed from the pulmonary side, there was no evidence suggesting an inflammatory or neoplastic origin. Thoracic surgeons know well that single, fluid-filled intrapulmonary bronchogenic cysts nearly always have such smooth, thin septa at the point of origin from a bronchus. Almost certainly these septa are congenital in origin.

Had the specimen herein described been examined by multiple section technique, the true situation might have been lost in a picture of multiple cysts of varying size. The pathological diagnosis would then have been multiple bronchogenic cysts, implying both a separation of the elements and also multiple bronchial obstructions. It is suggested that perhaps most, if not all, so-called multiple, intrapulmonary bronchial cysts are examples of bronchial mucoceles, and that if such diseased portions of lungs were examined by tracing the bronchial systems in a retrograde manner, the true situation would be recognized.

Every other bronchial cyst of the lung removed by the author has shown several small outpouchings in the large cyst wall. These are almost certainly dilated bronchial branches *partly* taken up in, and *partly* compressed by, the major cyst mass. It is probable that such an intrapulmonary bronchial cyst is no more than an advanced stage of a "mucocele of the lung."

True congenital bronchiectasis probably exists as another variant of a mucocele, originating by rupture of the obstructing septum after dilatation of the tributary bronchial system has taken place.

A study of the literature fails to reveal an instance where such obstruction of the bronchial system, and coexistent distention with secreted mucus, existed along with inflation-deflation phenomenon of the dependent alveoli, as was discovered in the case under discussion. To produce this combination of circumstance, either multiple obstructions were present at each alveolar duct entrance to prevent entry of mucus into the alveoli, or else (and more likely) the mucus was too viscous to pass through the minute openings. The air passage must have been transalveolar and transsegmental. The experiments of Loosli[3] are of pertinent interest in this regard. He produced bronchial obstruction in dogs by painting the bronchus with 35 per cent silver nitrate. As a result of this, a non-inflammatory atelectasis developed. The following observations were made: "In thick sections which afford a surface view of the alveolar walls, numerous openings or pores are seen in the intercapillary spaces. In thin sections, these appear as partial or complete interruptions in the alveolar septa." — "The small and terminal bronchioles, which do not possess cartilage in their walls, do not contain mucus and are collapsed. The cuboidal cell membrane is intact and thrown into folds. Some contain collections of macrophages, but usually they are empty." — "The alveolar ducts and alveoli appear as slits and crevices in what might be called 'solid tissue.'" These experiments in dogs produced a situation very similar to that of the lesion described. There was bronchial obstruction with the larger bronchi distended with mucus,

whereas the terminal bronchioles and alveoli were collapsed and free from mucus. It is probable that if the experiment were repeated in such a manner as to produce obstruction with the silver nitrate in a segmental bronchus, the larger bronchi would become filled with mucus as described above, but the alveolar portions and the terminal bronchioles would distend with air by way of collateral ventilation from the adjacent segments, thus representing an exact analogy to the case reported herein.

Thoracic surgeons are well aware that when segmental bronchi are clamped shut at surgery, positive pressure intratracheally, by the anesthetist, almost routinely produces inflation of the alveolar system of the occluded segment. This does not usually occur if there exists significant parenchymatous disease of that segment.

Baarsma, Dirken and Huizinga[4] have carried out experiments in man which well demonstrate the transsegmental, transalveolar respiration which exists in the absence of parenchymatous disease. They obstructed either the entire lower lobe bronchus, or the lower lobe bronchus just distal to one or more of the lobar segmental bronchi, resulting in obstruction of only some of the segmental bronchi of the lower lobe. They found that when the bronchus was completely obstructed, atelectasis occurred and there was no evidence of collateral ventilation; whereas, if only a side branch were obstructed leaving at least one of the lobar segmental bronchi unobstructed, then collateral ventilation did occur and atelectasis did not supervene.

The failure of the distended mucus-filled tributary bronchi to give significant x-ray shadows is of great interest. Presumably the failure is due to an unfavorable density contrast by the scattering of the pathology throughout distended lung of considerable depth. It indicates that an

TABLE I: PULMONARY CYSTS

1. MECHANICAL			
	BRONCHOGENIC	— Variants	— Mucocele
			Cyst
			Congenital Bronchiectasis
	PARENCHYMAL	— Variants	— Blebs
			Bullae
			Pneumatoceles
2. INFLAMMATORY			
Bacterial			
Fungal			
Parasitic			
3. NEOPLASTIC			
Benign			
Malignant			
4. ABERRANT			
Gastro-enterogenous, etc.			

individual may have an apparently normal chest roentgenogram followed subsequently by the appearance of a fluid-filled or air-filled cyst (as the process continues or results in rupture) and yet have a truly congenital disease. In other words, the late appearance of a cyst by x-ray does not necessarily mean the cyst is an acquired one.

Presumably the lack of pigmentation typical of congenital bronchogenic cysts is due to the small size of the intersegmental and transalveolar communications and not to the failure of air to enter the affected tissue. The tiny communications probably act as a selective filter, removing carbon particles. It is a common finding that the intersegmental planes bordering such an obstructed segment have an extraordinary deposit of pigment.

The aberrant drainage of a segmental bronchus from one lobe into a tributary of a segment of another lobe is of interest.

The above simple classification is offered because of its simplicity and its correlation with the clinical aspects of diagnosis. All subdivisions are self-explanatory, excepting the mechanical group. It is considered that there are two major varieties of mechanical cyst; (a) bronchogenic, and (b) parenchymal. Bronchogenic cysts arise by obstruction to a portion of the bronchial tree, usually because of a congenital septum or portion of atresia, but possibly also, on occasion, from an acquired obstruction. Three variants of the same fundamental lesion are: (1) mucocele, (2) cyst, and (3) congenital bronchiectasis, as discussed previously. Parenchymal cysts, likewise, are thought to arise from a mechanical interference with collateral ventilation. Such interference could be temporary or permanent, and probably results from pulmonary edema, inflammation, or fibrosis. The variants of the same pathology include blebs, bullae, and pneumatoceles, which are probably nothing more than different degrees of the same process.

A similar interference with collateral ventilation, but in such a manner as to produce the reverse effect; namely, the loss of air-filling rather than an increase in the amount of air trapped, might explain the areas of plate-like atelectasis frequently found during minor inflammation and/or edema of lower lobes.

SUMMARY

A case of mucocele of the lung has been described. It appears to provide the "missing link" between bronchial cysts and congenital bronchiectasis. It suggests that pulmonary blebs, bullae, pneumatoceles, and perhaps emphysema arise from disturbances in the collateral ventilation of the lungs.

RESUMEN

Se describe un caso de mucocele del pulmón. Parece que proporciona el "eslabón faltante" entre los quistes bronquiales y la bronquiectasia congénita. Esto sugiere que las ampollas, bulas, neumatoceles y tal vez el enfisema emanan de trastornos de la ventilación colateral de los pulmones.

RESUME

L'auteur décrit un cas de mycocèle du poumon. Cette affection semble tenir le juste milieu entre les kystes bronchiques et la bronchectasie con-

génitale. Elle amène à penser que les kystes aériques, les pneumatocèles et peut-être l'emphysème pourraient venir de perturbations de la ventilation collatérale des poumons.

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Temporary "Spontaneous" Paralysis of the Diaphragm*

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The published articles on spontaneous paralysis of the diaphragm, particularly in the English literature, are few indeed. This is remarkable because the condition may, at times, give rise to manifestations every bit as dramatic as coronary occlusion. Frequently, however, the symptoms are minimal or absent altogether.

Unilateral elevations of the diaphragm may be a normal finding. In most persons the hemidiaphragms are not at the same level and their relative position may change with normal functional processes, e.g. food intake, etc. If the upward displacement is marked and accompanied by impairment of function, it is due to one of the following causes:

1) It may simply be an upward displacement by "push" or "pull," i.e. either by retracting intrathoracic processes or by space-consuming intra-abdominal conditions. These displacements, as a rule, are not as marked as those caused by the other conditions listed, and the pathognomonic mechanism is self-evident.

2) Affections of the muscle itself will produce an elevation of the diaphragm. Attention was drawn to this myositis by Joannides, in 1946,[1] who first described a primary diaphragmitis which he named "Hedbloom's Syndrome," and reported 12 cases presenting this symptom-complex. Another 12 cases were reported by Meyler and Huizinga in 1950,[2] and in their patients the involvement was secondary to inflammatory processes in the abdomen and thorax.

3) Most frequently, elevations of the diaphragm are caused by lesions of the phrenic nerve. There is abundant literature on the anatomy and physiology of phrenic innervation, and the subject has been studied extensively since Schroeder described the effects of artificial interruption of the nerve in 1902.[3] Curiously enough, up to Schroeder's investigations, paralysis of the phrenic nerve was considered a rare and exceedingly serious, if not fatal, condition. The few cases which were reported leave ample doubt as to the correctness of the diagnosis. The dismal outlook attributed to the disease was due to the fact that it was usually a complication of a serious primary affliction.

In recent times most phrenic nerve paralyses are due to mechanical interruptions, either surgical or traumatic. Therapeutic severance of the nerve is still a valuable procedure in the treatment of tuberculosis and accounts for the overwhelming majority of cases encountered. Uninten-

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Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are a result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

tional damage to the phrenic nerve during operative procedures around the neck and in the thorax also furnished a fair number of cases.

Traumatic paralyses of the phrenic nerve do not appear to be as rare as was thought. Frequently they are due to birth-trauma and are accompanied by paralysis of the brachial plexus (Erb's palsy).[4-6] They would, without questions, be discovered more frequently if every newborn with brachial paralysis were x-rayed to ascertain the motility of the diaphragm. The obvious paralysis of the extremity in conjunction with the relative absence of symptoms in one-sided diaphragmatic paralysis commonly accounts for the phrenic lesion being overlooked. In rare instances, a lesion of the phrenic nerve will be found where there is no damage to the brachial plexus,[7-9] and occasionally both nervi phrenici may be involved.

While there are ample descriptions of these surgical-traumatic lesions "Medical," or as it is commonly called "Spontaneous" diaphragmatic paralysis is mentioned only in a very few investigations and case reports, and most of these are in foreign medical writings.[10-19]

The phrenic nerve contains motor and sensory fibers in the proportion of about two to one.[20] It arises chiefly from the fourth cervical nerve, but receives a branch from the third and another from the fifth. It descends across the front of the Scalenus anterior and beneath the Sternocleidomastoideus and passes in front of the first part of the subclavian artery, between it and the subcavian vein. Within the thorax, it descends nearly vertically in front of the root of the lung, and then between the pericardium and the mediastinal pleura to the diaphragm. The right nerve is situated more deeply, and is shorter and more vertical in direction than the left; it lies to the right of the innominate vein and superior vena cava. The left nerve is rather longer than the right, from the inclination of the heart to the left side, and from the diaphragm being lower on this than on the right side. In the superior mediastinal cavity it lies between the left common carotid and left subclavian arteries, and crosses superficially to the vagus on the left side of the arch of the aorta.[21]

A paralysis of the nerve may be due:

(1) To destruction of the nuclei.

(2) "Neurotoxic" infections or poisons.

(3) Encroachment on the nerve itself during its course, either by invasion or by compression.

The symptoms of phrenic paralysis may be completely absent, especially if the encroachment is slow or if the lesions are central. In those cases a paralyzed diaphragm will not be detected unless x-ray examination is made. This may account for the relatively large number of cases described in pulmonary tuberculosis, where routine radiography has been done for many years. In other cases where the paralysis is secondary, the symptoms, though present, are submerged in the generally much more impressive primary disease, and are apt to be overlooked by doctor and patient. If symptoms are manifested they tend to be fairly characteristic.

There will be a more or less sudden pain in the shoulder on the affected side. This pain occasionally may resemble the "anginal" pain-distribution,

though it is usually more localized at the tip of the shoulder on the affected side, along the upper border of the trapezius muscle and in the supraclavicular area. At the same time, an internal pain may be described anywhere within the affected side of the chest, and there is shortness of breath which is aggravated by acute anxiety.[20]

If paralysis of the hemidiaphragm is suspected, confirmation may be easily obtained by fluoroscopic examination. The involved diaphragm is elevated and immovable. These findings, however, are not sufficient for a diagnosis of interrupted diaphragmatic innervation since they may also be seen in diaphragmitis, subphrenic abscesses and other conditions. More pathognomonic are the so-called "paradoxic movements," i.e. opposite movements to those of the healthy side with deep respiration and still easier detected with sudden changes of intrathoracic pressure as in coughing and sniffing.

Infectious diseases may cause phrenic paralysis either by invasion of the central nervous system (e.g. poliomyelitis[14]) or by the production of neurotoxins (e.g. diphtheria[11]). Similarly, numerous organic and inorganic[16] nerve-toxins have been described as causing interruption of diaphragmatic innervation. Most commonly, however, the nerve is compressed, invaded or destroyed in its course through the thoracic cavity by neoplastic[10,14,18,19] in inflammatory processes.[11,15,17,18] An unexplained paralysed phrenic nerve, today, is most likely to be considered evidence of intrathoracic malignancy, although until only a few years ago it would have been most frequently thought to be due to tuberculosis. This was probably due to the fact that patients with tuberculosis were more frequently and regularly examined with x-rays than any other group of people. The best study of phrenic paralysis in tuberculosis was written by Gimeno Ondovilla,[17] who published his series of findings. Most of his patients, like most of the other published reports, had permanent paralyses, although one showed a "potentially reversible process," i.e. if this patient had lived, function might have reappeared. Temporary, "spontaneous" phrenic paralysis is extremely rare and it appears worthwhile reporting a case observed in this hospital.

Case Report

H.B., a 55 year old white male, who was well until the beginning of 1947, when he complained of anorexia, weight loss and unproductive, hacking cough. He was admitted to the Veterans Administration Hospital, Oakland, California, in March 1947, and pulmonary tuberculosis was diagnosed on the basis of acid fast bacilli in his sputa and lesions in both apices and the right infraclavicular region seen on x-ray films. These were read as exudative lesions and the lordotic view showed what appeared to be a small cavity in the left apex. He was transferred to Veterans Administration Hospital, Livermore, California, in September 1947. Pneumoperitoneum was induced in October 1947, and he was discharged to out-patient treatment, his pneumoperitoneum being refilled once a week. In July 1948, his sputa had remained negative for acid fast bacilli for more than six months. The x-ray film findings at that time were interpreted as bilateral, fibroid minimal disease. From June 1948, until February 1949, he felt well, had no complaints and led a moderately active life. Intensive questioning failed to reveal any exposure to

neuro-toxic substances of any sort. On February 5, 1949, he returned for a regular refill of his pneumoperitoneum, and at this visit he stated that for the preceeding several days he had noticed slight shortness of breath. He attributed this shortness of breath to excessive air in the abdominal cavity and requested that less air be given. This request was complied with. Two days later he developed a sudden sharp pain in the left chest. The pain was localized in the intra-scapular area and did not show any radiation. It occurred around three o'clock in the morning and woke him from a sound sleep. It was aggravated by respiratory movements and increased upon deep inspiration. It slowly and gradually subsided over the next 48 hours. On February 21, he was well on awakening and started to get up to go to the bathroom when he suddenly felt a severe pain, again in his left chest, and a marked shortness of breath. He returned to bed as quickly as he could and his symptoms eased somewhat, but when he tried to get up, the pain recurred. He consulted a local physician who advised immediate hospitalization. The patient was admitted around noon of the same day. Upon admission the patient appeared to be in rather acute distress, although the only significant finding was a tachycardia of 116, with a normal sinus rhythm. Examination of the lungs showed only some minimal change of breath sounds in both apices. Extensive work-up showed essentially normal laboratory findings save for a slightly elevated sedimentation rate (21 mm./hr.), and acid fast bacilli in several sputum specimens examined. An electrocardiogram was normal. Posterior-anterior x-ray films of the chest were interpreted as showing fibrotic and infiltrative lesions in both apices without any cavity being found. Pneumoperitoneum, displacing both diaphragmatic leaves upward about equally, was present. The patient was put on bedrest and no specific therapy was instituted. He appeared to be improving, had no complaints. No further refills of his pneumoperitoneum were given.

By the middle of March the patient felt well and was semi-ambulatory. Repeated follow-up examinations by physical, fluoroscopic and electrocardiographic means did not produce any new findings.

On March 23, 1949, the patient developed a low grade temperature between 99.6 and 101.0 degrees F. There were no localized complaints nor any new findings. On March 28, around 10:30 p.m., while resting quietly in bed, the patient suddenly developed a severe pain in the right side which was localized anteriorly and radiated into the region of the right shoulder tip, the lateral aspect of the supraclavicular and trapezius regions. This pain was accompanied by severe dyspnea, grunting, and polypnea of 43 respirations per minute. His face was flushed, his pulse rapid (125 per minute), and he appeared apprehensive and fretful. Fluoroscopic examination of his chest was done immediately and revealed pneumoperitoneum to be still present though no refill had been given for six weeks prior to this episode, the right diaphragm being elevated four centimeters higher than the left one. No active movement of the right diaphragm was visible but paradoxic movements could be observed on sniffing. His temperature went to above 103 degrees F. orally and the patient was obviously and severely ill. He was put to bed and treated with sedation and intermittent oxygen. During the following four days his temperature remained elevated. Physical condition and fluoroscopic and roentgenographic findings remained unchanged. His sputa became slightly blood tinged and upon physical examination there was a decided lag of respiration on the right side with decreased resonance especially over the lower part. Numerous moist rales and areas of bronchial breathing in the paravertebral region at the base were found. All these symptoms slowly and gradually subsided within one week. Sputum examination for malignant cells during that time gave negative results. After the acute symptoms subsided bronchoscopy was performed. A normal bronchial tree was found on the left side. At the level of the orifice of the right upper lobe several irregular cartilage-hard nodules were seen protruding into the lumen from the lateral wall. Similar, but less marked, changes were seen on the wall slightly below the orifice of the upper lobe. Several small biopsies were taken

from these regions because on bronchoscopic examination they suggested carcinoma. However, pathological examination of these bite-specimens revealed only inflammatory non-specific lesions. The mucous membrane was intact and only small round cells were seen in moderate numbers. The patient continued to improve subjectively, and objectively the findings of compression in the lower lobe had disappeared by the 15th of April. On April 20, the patient was fluoroscoped again and a normally movable diaphragm was found. This was confirmed by a roentgenogram. From then on there was a steady improvement of all subjective and objective signs and symptoms. The patient remained well; there were no acid fast bacilli in his sputa. The x-ray films remained unchanged and during a two year observation period no recurrence of the paralysis was discovered. The patient was discharged from hospital care in January 1950, but now (September 1951), is still under out-patient observation at this hospital.

SUMMARY

This patient with an established diagnosis of pulmonary tuberculosis first developed what in retrospect appears to have been attacks of bilateral phrenic neuritis, and then phrenic paralysis, lasting less than three weeks. Although it is impossible to prove a definite diagnosis, the pre-existing tuberculosis and the absence of other significant findings, together with the bronchoscopic observations suggest a tuberculous process, probably of the lymph-nodes compressing the phrenic nerve, as the most likely cause.

A review of the causes of unilateral diaphragmatic elevation is presented in connection with this report.

RESUMEN

Este enfermo con un diagnóstico establecido de tuberculosis pulmonar presentó lo que retrospectivamente parece haber sido ataque bilateral de neuritis frénica y después parálisis frénica, con menos de tres semanas de duración. Aunque es imposible demostrar un diagnóstico preciso, la tuberculosis preexistente y la ausencia de otros signos importantes, así como la observación broncoscópica, sugieren que se trataba de un proceso tuberculoso y que la causa más probable fué la compresión producida por los ganglios linfáticos.

Una revisión de las causas de elevación diafragmática unilateral se presenta en relación con este trabajo.

RESUME

Il s'agit d'un malade atteint de tuberculose pulmonaire. Il présenta ultérieurement des manifestations qui, rétrospectivement, semblent avoir été une atteinte de névrite bilatérale du nerf phrénique, suivie de paralysie phrénique ayant duré moins de trois semaines. Il est impossible dans ce cas d'apporter un diagnostic certain; cependant l'auteur pense qu'il s'agit d'un processus tuberculeux probablement ganglionnaire, ayant comprimé le nerf phrénique. En faveur de cette hypothèse, il note la tuberculose préexistante, l'absence de toute autre manifestation pathologique et les constatations bronchoscopiques.

Un exposé des causes habituelles de l'élévation unilatérale du diaphragme est présenté à l'occasion de cette communication.

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Report of the Committee on Chest Diseases in Institutions*

American College of Chest Physicians

Authorities in the field of chest diseases have long recognized the problem of tuberculosis in hospitals for the mentally ill and penal institutions. In 1898, the Tenth Annual Report of the Board of Trustees and Officers of the Minnesota Hospitals for the Insane contained the following statement: "Experience has demonstrated that in a hospital for the insane that dread disease, consumption, is quite prevalent. We would strongly recommend the erection of a small detached ward for patients already afflicted."[1] In 1904, the Congressional Record incorporated an article on "Tuberculosis in Penal Institutions."[2] The problem of prisoners' health in general received increasing attention, and by 1929 every state and federal prison was visited by a physician for the National Society of Penal Information.[3]

However, not until 1946, when the American College of Chest Physicians' Committee on Chest Diseases in Mental and Penal Institutions was established, was any persistent nation-wide attempt made to standardize and modernize the methods of caring for the tuberculous confined to mental and penal institutions. Since then, the literature on tuberculosis in institutions has been growing, and interest in the subject has broadened. During the past year, a considerable number of papers and reports have been published, reflecting a positive approach to the problem.

Prisons have had practical difficulties in undertaking tuberculosis control programs. The training of personnel who staff the most progressive of such institutions is directed toward reconditioning the inmate's attitude to prepare him to return to society as a responsible citizen. Therefore, even in the best and most modern prisons, caring for physical illness is likely to take a secondary place in prison activities. The authorities—generally unfamiliar with medical problems—may not fully understand the need for tuberculosis control and, preoccupied with the social problems which are their first concern, may lack interest in establishing tuberculosis control programs within the walls of their institutions.

Tuberculosis control in institutions for the mentally ill, on the other hand, has had the advantage of medically oriented staffs. Although, of course, the emphasis in such hospitals is on psychiatric treatment, the medical training of the physicians in charge makes them more readily understand the value of early case finding, isolation, and treatment in a disease like tuberculosis. Always plagued by insufficient medical and nursing help, they have regarded with reluctance the introduction of any new procedure, such as tuberculosis control, as an additional burden for the already overworked staff. There were some instances during the late 1930's

*Presented November 21, 1952, at the dedication of the Herbert A. Burns Memorial Hospital of the Anoka State Hospital, State Division of Public Institutions, Anoka, Minnesota.

and the early 1940's of institutional tuberculosis control activities in various states. Minnesota, New York, Illinois, and California set up energetic programs; and no doubt there were others which were not reported and thus did not receive wide attention. But in recent years, authorities have recognized the threat of tuberculosis in their populations and are beginning to take aggressive action against it, in spite of the special problems it poses in their situation. It is timely that the fine work being done in this respect in the Neuropsychiatric Division of the Veterans Administration be emphasized.

In 1936, Illinois' Governor Henry Horner appointed a committee of physicians to investigate and report on health conditions in the State's various penitentiaries. Among the recommendation submitted by this committee was the establishment of tuberculosis control activities.[4] The first four years (1939-1943) of work in Illinois was called to the attention of the American College of Chest Physicians and the American Trudeau Society. Part of this report dealt with the general lack of information which existed regarding institutional tuberculosis control conditions in other states. As a result of this report, which was given in 1944[5] on tuberculosis in prisons rather than in mental hospitals, the American College of Chest Physicians established the Committee on Chest Diseases in Penal and Mental Institutions in April, 1946. The American Trudeau Society set up a somewhat similar committee for a short period and presented an excellent report.[6] The interest of the American College of Chest Physicians' Committee was broadened later to include other institutions (state soldier and sailor homes, schools for the deaf and blind and crippled, etc.), and its name was changed to the Committee on Chest Diseases in Institutions.

The main objective of this Committee has been to encourage the establishment and maintenance of good tuberculosis case finding, isolation, and treatment in institutions. It should be stressed that there are important by-products of case finding, especially carcinoma of the lung and certain forms of cardiac disease. To accomplish this main objective, a national survey of existing tuberculosis control programs in institutions was under-

TABLE I: PERCENTAGE OF INSTITUTIONS REPORTING THE USE OF SPECIFIED PROCEDURES

Procedure	Mental (241)			Correctional (116)			Others (32)		
	Yes	No	Not Stated	Yes	No	Not Stated	Yes	No	Not Stated
Case Finding									
X-ray every new admission	78.4	21.2	0.4	59.5	38.8	1.7	56.2	43.8	—
Periodic chest x-ray films	87.1	10.8	2.1	82.7	14.7	2.6	68.8	28.1	3.1
Routine tuberculin test	20.3	76.8	2.9	24.1	75.0	0.9	53.1	43.8	3.1
Pre-employment chest film	74.3	24.9	0.8	68.1	28.4	3.5	56.3	28.1	15.6
Case Handling									
Have isolation tuberc. unit	66.8	21.2	12.0	33.6	48.3	18.1	37.5	62.5	—
Employ chest specialist	48.1	40.3	11.6	26.7	48.3	25.0	37.5	62.5	—

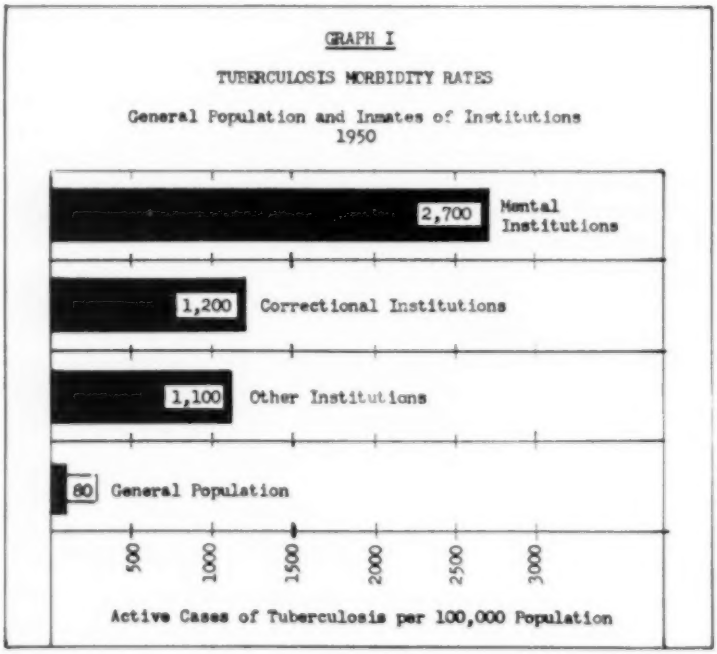
taken in 1951, and a five-page questionnaire was sent to each state, the District of Columbia, Alaska, Hawaii, Guam, and Puerto Rico. Among the information which this survey sought were facts on: case finding programs, morbidity and mortality from tuberculosis for the past 12 years; evaluation of diagnostic facilities; type of care administered, including collapse therapy

TABLE II: ACTIVE CASES OF TUBERCULOSIS AND DEATHS FROM TUBERCULOSIS AMONG INMATES OF INSTITUTIONS REPORTING

Active Cases				*Deaths**			
Institutions reporting on active cases	Population reported	Cases reported	Case rate per 100,000 population	Institutions reporting on deaths	Population reported	Deaths reported	Death rate per 100,000 population
Mental:							
231	511,064	13,911	2,700	206	465,746	1,946	418
Correctional:							
90	60,556	749	1,200	98	59,899	70	117
Other:							
27	27,224	287	1,100	27	27,224	40	147
Total† All Types Reporting:							
348	598,844	14,947	2,500	369	604,387	2,229	369

* Based on last year reported in the questionnaire (usually 1950).

† Figures shown by type of institution do not add up to the total, which includes figures for institutions where type was not reported.

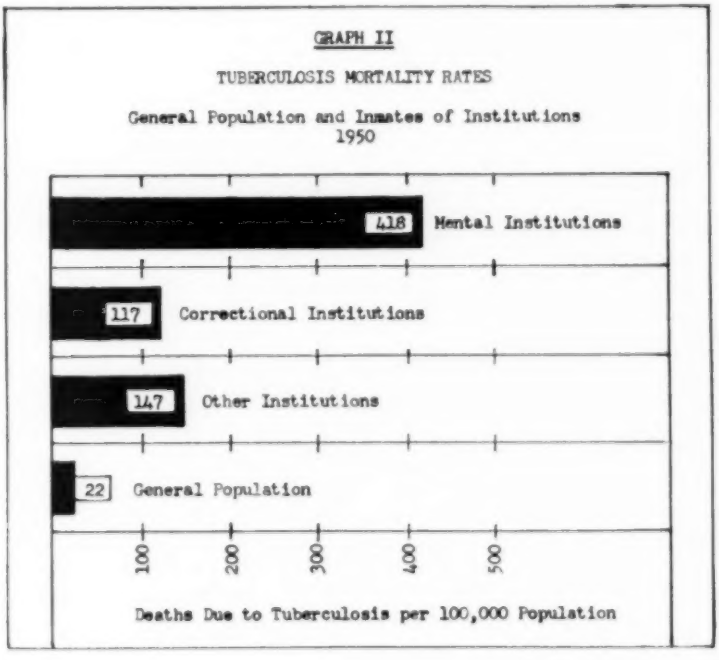


and chemotherapy; ratios of nurses to patients and physicians to patients; types of physicians giving care (chest physicians, general practitioners or psychiatrists); use of BCG; and rehabilitation services.

An analysis of the questionnaires indicates that institutions throughout the country do not have a quality of tuberculosis control which would be acceptable elsewhere in the same communities. While there is much to be desired in the case finding activities in many mental hospitals and prisons, there is even greater inadequacy in hospitalization and treatment of the suspected and known cases of tuberculosis. In many instances, there is no coordination between case finding and treatment, and little attempt is made to isolate cases discovered.

In many states, however, the trend toward the establishment of adequate, workable, tuberculosis control programs in state institutions is well under way. Ohio, New Jersey, Indiana, Pennsylvania, Louisiana, and Arizona have asked the American College of Chest Physicians' Committee for assistance in setting up acceptable tuberculosis control programs for their respective institutions. Wisconsin has surveyed its facilities, and a report will soon be made in the state medical journal. Examples of action in other states recently have been reported.[7]

The Committee is very familiar with the program carried out in Illinois and would, therefore, like to call attention to its progress. The institutional tuberculosis control program in the Illinois Department of Public Welfare covers 13 hospitals for the mentally ill and 12 educational and correctional



institutions (schools for the blind, the deaf, etc.; boys' and girls' training schools). The total population of 49,000 persons in these institutions have routine entrance and semi-annual chest films. The incidence of reinfection tuberculosis has decreased approximately 82 per cent in the period between 1945 and 1951. During essentially the same period of time, the number of deaths decreased 49 per cent.*[5]

Any mental hospital patient with questionable x-ray findings is placed in an observation ward for complete clinical work-up. Patients with active tuberculosis are isolated in separate facilities in each of the 13 units. Therapy in the Illinois institutions follows the accepted methods, including bed rest, antibiotics, and minor collapse procedures. Facilities for thoracic surgery have been and are being established.

Only a few inmates in the educational and correctional institutions have been detected with tuberculosis. These are routinely transferred to public sanatoria or, occasionally, are isolated within the hospital facilities of the institutions. Some 11,000 institutional employees of the Illinois Department of Public Welfare, under whose jurisdiction the 25 institutions fall, are included in the case finding program, and personnel who contract the disease are referred to public or private agencies for follow-up and care.

The Illinois Penal System, with approximately 8,000 inmates and 1,000 employees, is a part of the Illinois Department of Public Safety and has a somewhat similar control program. A geographically centralized tuberculosis hospital houses all inmates with active tuberculosis.

The high prevalence of tuberculosis in jails in the larger metropolitan areas has been stressed recently in Los Angeles, Seattle, and Chicago. Seventeen per cent of a group of alcoholics studied in Seattle revealed active pulmonary tuberculosis.[9] While metropolitan jails may not have as great a total inmate load as state penitentiaries, they at times annually incarcerate many more inmates than do state penitentiaries. The City of Chicago has over 4,000 inmates in the combined population of the House of Correction and the Cook County Jail and annually admit over 30,000 inmates. The Illinois State Penitentiaries admitted less than 4,000 inmates during the year 1951.

We have now reached the peculiar position in Illinois where patients entering state mental or penal institutions stand a far better chance of having their tuberculosis diagnosed early than do those admitted to the average general hospital. This paradox is the result of routine chest screening examinations, at least annually, in all state institutions and the present lack of this procedure in most of the general hospitals. This deficiency, however, is being corrected.

There are a number of agencies and organizations throughout the country which are concerned with the problem of tuberculosis in institutions. The American College of Chest Physicians' Committee on Chest Diseases in Institutions has attempted to coordinate its activities with groups which have similar interests. It was represented on the medical programs of the

*Statistics for 1952 show a 75 per cent reduction in the death rate since 1945.

American Prison Association meetings in Boston in 1948, in Milwaukee in 1949, in St. Louis in 1950, and again in Biloxi in 1951. The Group for the Advancement of Psychiatry, through its Committee on Hospitals, is preparing an excellent and comprehensive booklet, "The Tuberculous Psychotic Patient," setting forth the principles for tuberculosis control in the psychiatric hospital. The Veterans Administration, the American Trudeau Society, and the American College of Chest Physicians were called upon for consultation with the Group for the Advancement of Psychiatry's Committee on Hospitals in planning the booklet.

We have made a beginning in our approach to the problem of tuberculosis in institutions for the mentally ill, prisons, etc. With the combined efforts of interested organizations, the control of the disease in these settings can be accomplished. Of course, much is yet to be done. But we know that there is basically little or no difference in the therapy of tuberculosis of the general patients compared with those patients with psychosis or those incarcerated in penal institutions. There is a need for the establishment of broad basic standards to provide for the health of patients and inmates in institutions, and adequate staff and facilities for treating tuberculous patients must be provided if the disease is to be eradicated within these institutions.

The progress that has been made in the past, however, has not waited upon the formalization of standards nor the provision of adequate facilities. Many fine accomplishments have materialized in terms of existing situations which showed little promise at the beginning. Opportunities are presented to evaluate the influence of emotional status on inflammatory and other chest diseases. It is indeed satisfying to note the acceleration of progress toward the fulfillment of ideas for tuberculosis control in institutions.

Otto L. Bettag, Chicago, Illinois, *Chairman*
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Robert B. Kerr, Manchester, New Hampshire
A. A. Leonidoff, Poughkeepsie, New York
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Lloyd K. Swasey, Phoenix, Arizona.

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This report was presented at the dedication of the Herbert A. Burns Memorial Hospital, the diagnostic and treatment center for the tuberculous psychotic, for the Division of Public Institutions for the State of Minnesota. The achievements of Dr. Burns, a fellow Committee member, a great physician, epidemiologist, educator, and benefactor of humanity, were summarized in April, 1950.[10] His pioneering work in the eradication of tuberculosis together with the close cooperation of Dr. Edmund W. Miller,[11] Superintendent of the Anoka State Hospital, made an adequate center available at this hospital, less than 20 miles from the University of Minnesota School of Medicine. The nation and the world look toward Minnesota for leadership in tuberculosis control. The wisdom of Dr. J. Arthur Myers, then President of the American College of Chest Physicians, in his appointment of this Committee has borne fruit. It is not by coincidence that from Minnesota came Drs. Robert J. Anderson, Herman E. Hilleboe, James E. Perkins, and many other leaders.

The splendid assistance of the Division of Chronic Disease and Tuberculosis of the United States Public Health Service in presenting the above is gratefully acknowledged.

Otto L. Bettag.

Annual Meeting and Election of Officers

The 19th Annual Meeting of the College, recently held in New York City, surpassed all previous records for attendance at both scientific sessions and social functions. Approximately 1,500 were registered, including 1,200 physicians and approximately 300 non-physician guests. The previous registration record was attained in Chicago last year when 1,040 physicians and guests registered. There were 22 technical exhibits as well as 3 scientific and educational exhibits. At the President's Banquet held on Saturday evening, May 30, the capacity of the Grand Ballroom at the Hotel New Yorker was overtaxed.

The following officers, Regents, and Governors were elected:

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Washington: Byron F. Francis, Seattle
West Virginia: George R. Maxwell, Morgantown
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U. S. Navy: Comdr. Sidney A. Britten, Washington, D. C.
U. S. Public Health Service: Robert J. Anderson, Washington, D. C.
U. S. Veterans Administration: Roy A. Wolford, Washington, D. C.
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Eastern Provinces: J. J. Quinlan, Kentville, N. S.
Pacific Provinces: W. Elliott Harrison, Vancouver, B. C.
Western Provinces: Leslie Mullen, Calgary, Alberta

ALVIS EUGENE GREER

President

AMERICAN COLLEGE OF CHEST PHYSICIANS

1953 - 1954

President's Address

The Rock On Which We Stand

ALVIS E. GREER, M.D., F.C.C.P.
Houston, Texas

My friends and associates of the American College of Chest Physicians, you have given me high honor and have invested within me a deep sense of duty and obligation to you and to the suffering peoples of this world. Pray believe me, I have no personal feeling of conceit in attaining this high office. I am burdened down with a knowledge of my own mediocrity. The glory lies within the men preceding me including Mr. Murray Kornfeld and Dr. Charles M. Hendricks who conceived and reared this organization. To such men goes all the honor. I accept the trust bestowed upon me with modesty and humility, being forewarned of my inadequacies but trusting that such consciousness will serve me well in charting my course. With your help and as God wills, I promise to keep that trust.

The privilege of speaking briefly of the world-wide problems besetting us is one I cherish and will keep tucked away in my heart for my remaining days. Our calling is so closely aligned with the ministry of God that the two are inseparable. Upon the mantles of both the medical profession and religion have fallen the harsh imprints of selfish, greedy, ambitious, godless men. I shall talk plainly of such malign attempts to take from us the inherent, traditional liberties of Man, and give you my ideas dealing with our course of action to preserve our future.

A primary conviction—a principle if you please—is that it is unnatural and godless to seek selfish gain, bounded by territorial limitations, and forget we are first of all men with the same God, a similar objective, a like fate. To forget such a deep principle, while adhering strictly to state boundaries, would produce lopsidedness, the most perilous disease in the world, and certainly plunge us into Stygian darkness and eventual failure.

An adherence to the laws of Nature and Nature's God is necessary to achieve our true objectives as healers and comforters of Man. It is essential to remember this truism—the unclean cannot cleanse the unclean. Men throughout the world are groping in the dark as though in a dense fog stretching out their hands into the unknown. They do not seem to realize the fog may hide things but not destroy them; they are borne aimlessly like driftwood upon a raging sea. We have become both the victims and the tools, tossed about like navigators without goal or compass. The world is filled with babblers voicing opinions as light as thistledown and their predictions will fall to earth like autumn leaves. The fault lies within us—we are the majority—they the minority. We, as men and nations, should expect adversity; it is in such a way we may be purified and strengthened. Mutual friendship is our refuge in adversity. We must have faith in one another and realize the future begins with the present. We must keep faith alive by constantly rekindling, realizing when it is extinguished, it may not be lighted easily again. We must probe our minds and hearts for our weaknesses, for as a chain is as strong as its weakest link, so our strength may be gauged by our inactivity and impotence. Friendship, faith and hope can endure only upon the burning altar of religion based on God—not upon creed alone.

We are beset by many difficult problems, economical and political, whilst the sword of Damocles hangs over us. The tyrants, despots, the free-thinkers strive to out-Herod Herod in changing the traditional principles of life for the political expediencies of the moment. In such a way precedents are born and unhappily

through continued use, unthinking men may come to think of them as principles. Disraeli once said, "Precedent is the shroud of principle, and embalms a principle." Truer words were never uttered. We need not be concerned about the dangers to our Christian way of life by the Hitlers, Stalins or Malenkovs of this or any other age. The minds—the souls—of free men given time, sooner or later, feel repulsion at such gross error. The traditional belief in the power of God and prayer lies too deeply in the minds and hearts of men to become subservient to such patently evil and godless ideologies. The real danger lies within the invisible shadows of the shrewd, powerful forces amongst us, walking in our midst with seeming benignity and harmlessness. They are the breeders of discontent, mischievous, evil harbingers, dissociating creed against creed, race against race, color against color and they may be found among the lowest and highest of us. Was not Judas Iscariot among Christ's twelve chosen disciples? These are the levelers who wish to level everyone down as far as themselves but cannot bear to raise themselves to a higher status of life. Jesus said, while instructing and cautioning his disciples, "And a man's foes shall be they of his own household." The entire world is our household, my fellow physicians, and we as citizens of the world, can aid materially by our example in bringing peace and love to mankind. Most men are good—at least they have within themselves inherent convictions of God and righteousness. It is only when an evil minority influences them they may become subhuman in thought and tendencies. The reason for this I do not know. It is not for me to know all of God's plan. My thesis is for the present and the future, whatever the trials may be. I can only hope and pray with faith in His wisdom that right will prevail.

Our chief peril is that an inferior minority will gang up and suppress a superior minority—superior by their adherence to God's precepts. You may have to bow to man-made laws, false ideologies may enslave you, whether they be fascism, communism, intense nationalism or democracy. Even democracy may do lip service to individual freedom, debased by power-hungry men or the unsound, irrational vagaries of so-called idealists; fanciful wanderings into the unknown of minds, educated though unintelligent. The sharp scalpel of the former group will do small harm compared to the dissecting needle of the latter, which will pry apart the substance of truth, liberty and traditional convictions of man and God into small threads, lying separate, ununited, cold and dead. It is in this way liberty may be nibbled away, piece by piece, part by part, and lost for ages. The present trend of our current free-thinkers to believe that happiness, social welfare, economic security and world peace can be adjusted solely by laws and regulations is fallacious. We have been beset for the past two decades with the stupid outpourings of such pettifoggery. I wonder if Sterne was correct when he said, "Free-thinkers are generally those who never think at all." For the mind of a free-thinker has no true vision; it is like the pupil of the eye; the more light pouring into it, the more it will contract. I presume many of us have the same thought; at least we might say truthfully they have a rather preponderant habit of thinking without logic and discernment.

When we realize that political questions are social problems it is evident they are religious as well. For is not the basis of all social pursuits a religious one? Certainly nothing is politically expedient which is morally wrong. A turning backward of decency and morality by dissident groups has been rife in this world for several decades. It is pitiable to consider that millions of God's children have been dominated by such godless ideologies. Fearful though their physical sufferings have been—and because of this we grieve with all our hearts—the greatest damage has been to their minds and souls. It is terrible to consider their broken bodies but the fact they have become indoctrinated with a malign ideology is infinitely worse because modern man has found a new method to destroy their souls. The false prophets amidst us, whether they be free-thinkers, or men seeking power, will be the ones who will spread such malevolence throughout the world.

Without such groups, tyrants like Hitler and Stalin would face a hopeless task because their influence would be of necessity limited—not world-wide. The infiltrators, sanctimonious and hypocritical, are the parasites, pathogenic and lethal, ever ready to prey upon the weak, ignorant, starving and underprivileged to attain their treacherous goal. It has been my observation that these pretending "do-gooders" frequently come from certain elements of the upper intellectual levels of our race—all too often men of formal education, sociologists, lawyers, politicians, educators, actors, statesmen, ministers, and—yes—physicians too. Although many of these individuals are honest and their convictions true to their lights, it is my opinion they are not intelligent in the truest interpretation of the word. Webster's dictionary defines an intellectual as "a member of a social class claiming to represent intellectual opinion"; whereas, an intelligent person is one "possessed of a high degree of understanding; a person of understanding." The mistaken idea of going backward, away from and denying God and his precepts is wrong—wholly, totally wrong—whatever the insidious infiltration of social levelers may falsely do or say. Ruskin has said, "Anything that makes religion a second object makes it no object—he who offers God a second place offers him no place." My friends, we cannot pick and cut away, slicing our traditional ways of right and wrong into little bits, without a ruinous end to all which is good and fair in life. Our future begins with our present. We must not be like men sitting alongside the stern lights of a ship, illuminating only the path it has passed. We must not go backward for it is vital that we go forward from where we now are.

Competition for advancement in general knowledge and science and to raise oneself socially and economically is praiseworthy. It must however be conducted in a human and sportsmanlike manner. The process that has made men human by selection of individuals with superior intelligence cannot cease now without cessation of advancement of the race. Ambition to strive and advance is laudable when based upon true humility, indicating we have a low opinion of our present and it is necessary to advance and improve ourselves. The mere possession of superior intelligence does not make all men good. What man among a superior group can stand up and say, "we are *all good?*" The group would be small numerically, indeed. Christ in His band of twelve chosen disciples found one betrayer—Judas Iscariot.

Murder, thievery, rapaciousness are bad—very bad—but what of covetness, arrogance, snobbery, falsification, evil scheming, greed, lack of tolerance, sympathy, understanding and of true humility. Lacking one of these, are we good—or evil? This is not to indicate that the bad in man predominates. Thank God such is not the case. But the hearing of good, whatever be the source, or the reading thereof, does not benefit a man unless he thinks and contemplates on that which is spoken or written. The latter course should give knowledge and perhaps promote wisdom. In such a manner one may know oneself, step by step, piece by piece, until the fortunate person may, in this way, envision his whole being both the good and evil. My thesis is that the possession of superior intellect does not fortify us from evil within our ranks. True virtue is the only nobility a man possesses whatever his race, creed or color may be.

What can we, as physicians, do to bring peace, happiness and adherence to the ineffaceable principles of life as laid down by Nature and Nature's God? We must have faith and be unafraid, working together not for selfish gain, but in the interests of Man, unholding the sacred tenets of our forefathers and the Bible. Jesus said to his disciples, "And fear not them which kill the body, but are not able to kill the soul." This is the rock on which we must stand. There is little natural enmity among the races of mankind except as a result of artificial organization by national leadership. The normal tendency of all species of animals is to befriend, protect and preserve the members of its own species. It is almost unknown among subhuman animals like the gibbon to attempt to exterminate

their own species; it remains for man, the superior species, to be alone in this respect.

We are creatures of God—men chosen as superior beings—and He has given us the means to use in preserving our inherent liberties, buffetted though we be by the storms created by evil men. A casual, disinterested attitude will not be adequate; action based on the teachings of Jesus Christ and a firm stand on them will suffice. Carlyle has said, "Religion cannot pass away. The burning of a little straw may hide the sky, but the stars are there, and will reappear." Our faith in one another and in God should be simple and unbending. The duty of a true physician is to be a healer and restorer of men. We must shed the light of our goodness, brotherhood and virtue throughout this disordered world, be ever conscious that "as the earth needs the sun in order that the gardens may bloom and the trees lift up their great branches, so men need the presence of God in order that the flowers of their virtues may grow and blossom."

My friends, such is the promise of Nature and Nature's God to each and all of us. May He speed and bless you throughout life, according to the fruits you bear unto Him.

BIOGRAPHY

ALVIS E. GREER

Dr. Alvis E. Greer was born in Equality, Ill., May 6, 1885. He graduated with the degree of Doctor of Medicine from Northwestern Medical School in 1908 and attended the Harvard Postgraduate Medical School. He is the author of numerous articles in medical literature including the chapter on "Fungus Diseases of the Lung" in the book, *Non-Tuberculous Diseases of the Chest* now in press.

Doctor Greer is a Fellow of the American College of Chest Physicians; Professor of Clinical Medicine, Baylor University College of Medicine; Director of the Greer Clinic, Houston, Texas; Diplomat, American Board of Internal Medicine; Director of the Common Cold Foundation; visiting physician, Hermann, Methodist and St. Joseph's Hospitals, Houston; and consultant, Jefferson Davis Hospital and Houston Tuberculosis Hospital.

He has served the College as Regent for Texas, a member of the Executive Council, and Chairman of the Financial Section of the Council on Research.

Doctor Greer and his wife, Maida Davis Greer, have three children—June, Dorothy, and Thornton.

MEDICAL SERVICE BUREAU

POSITION WANTED

General surgeon, male, age 43, Lithuanian, former tuberculosis patient, seeks position in sanatorium, no family, state license pending. Please address all inquiries to Box 278B, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

POSITIONS AVAILABLE

Staff positions: open at state tuberculosis sanatorium in Pacific Northwest for general or special physicians. Give full qualifications, training, etc. Address Box 265A, American College of Chest Physicians, 112 East Chestnut St., Chicago 11, Ill.

Staff physician wanted for approved county tuberculosis hospital, American citizenship and Ohio license necessary. Attractive salary. Please address all inquiries to Box 266A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Medical director wanter for 100-bed unusually active tuberculosis hospital in southern Illinois. Sanitarium presently staffed by surgeon but has a vacancy for a medical director and a resident. Institution two years old and extremely well-equipped. Please address all inquiries to Box 267A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Thoracic surgery resident: residency approved by Board of Thoracic Surgery and Council on Medical Education and Hospitals. Salary $300 per month, plus maintenance. Require U. S. A. citizen, eligible Ohio license. Please address all inquiries to Box 268A, American College of Chest Physicians, 112 East Chestnut Street, Shicago 11, Illinois.

Wanted immediately: superintendent of tuberculosis hospital, 65 beds. House and complete maintenance for self and family. Salary $7080-$8580. Apply Hampshire County Commissioners, Courthouse, Northampton, Massachusetts.

Assistant Medical Director wanted for 225 bed sanatorium in Birmingham, Alabama. For details write Arthur J. Viehman, M.D., Superintendent, Jefferson Tuberculosis Sanatorium, Birmingham 9, Alabama.

Resident wanted for 70 bed tuberculosis hospital located thirty miles west of Chicago. Apply to D. B. Radner, M.D., Medical Director, Winfield Hospital, Winfield, Illinois.

Staff Physician wanted for new 120 bed tuberculosis hospital, 28 miles from San Francisco. Salary scale: $649 to $811 per month. County Civil Service tenure; prospectus available after July 1. California license necessary. Inquire, Superintendent, San Mateo County Sanatorium, 200 Edmonds Road, Redwood City, California.

COLLEGE EVENTS

NATIONAL AND INTERNATIONAL MEETINGS

Interim Session, Semi-Annual Meeting, Board of Regents,
St. Louis, Missouri, November 29-30, 1953.

20th Annual Meeting, American College of Chest Physicians,
San Francisco, California, June 17-20, 1954.

Third International Congress on Diseases of the Chest
American College of Chest Physicians
Barcelona, Spain, Fall, 1954

POSTGRADUATE COURSES

8th Annual Postgraduate Course on Diseases of the Chest,
Knickerbocker Hotel, Chicago, Illinois, September 28 - October 2, 1953.

6th Annual Postgraduate Course on Diseases of the Chest,
Hotel New Yorker, New York City, November 2-6, 1953.

Announcements

The isolation of pure adrenocorticotrophic hormone (ACTH), developed by a team of half a dozen chemists in the Armour Laboratories division of Armour and Company has been announced. ACTH, the pituitary hormone which stimulates the growth and function of the adrenal cortex, was first introduced for medical use by the Armour Laboratories in 1949. Since then, it has revolutionized the treatment of many complex diseases such as arthritis, asthma, allergy, rheumatic fever, nephrosis, and others. Because its therapeutic effects are exactly the same as Armour's currently marketed ACTH, the pure product will not be put on the market but will be made available for research. Armour Laboratories also announce a new and simple method of clearing throat and bronchial passages in respiratory diseases. The treatment is based on the use of trypsin, a digestive enzyme, in the form of an aerosol, or fine mist. It is recommended for use in bronchial asthma, acute and chronic purulent bronchitis, emphysema, atelectasis, bronchiectasis, pneumonitis, and tuberculosis. Tryptar Aerosol is not a cure in any of these diseases, but it brings relief with as little as one treatment.

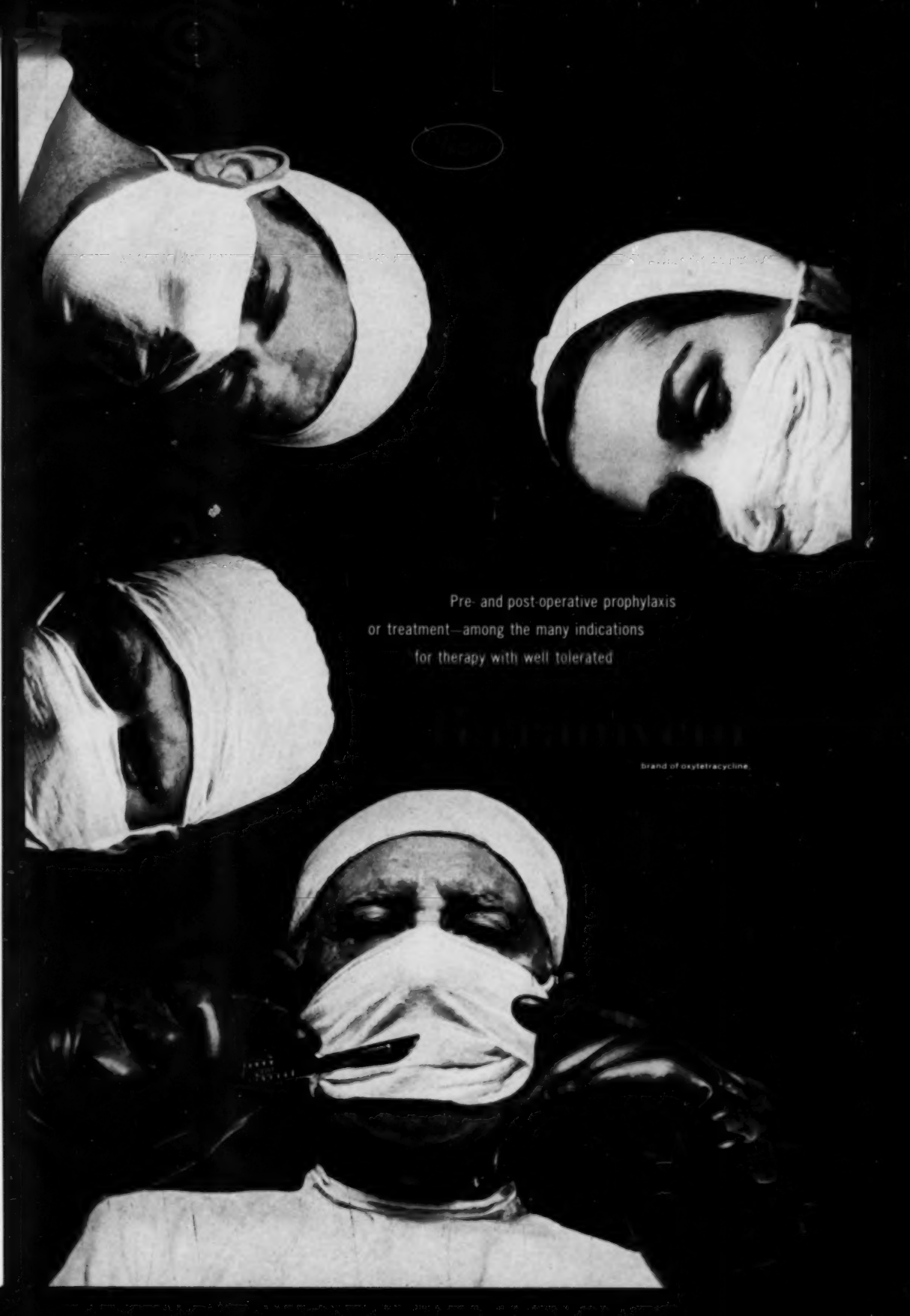
The Federal Communications Commission issued the following statement: "The operation in the industrial, scientific and medical service, of medical diathermy equipment, industrial heating equipment, and miscellaneous equipment of a type which emits radio frequency energy upon frequencies within the radio spectrum constitutes a serious source of interference to authorized radio services operating upon the channels of interstate and foreign communication unless precautions are taken which will prevent the creation of any substantial amount of such interference." A low cost shielded enclosure specifically designed for the purpose of erradicating this problem has been announced by the Ace Engineering and Machine Company of Philadelphia.

The first intramuscular digitoxin for safe predictable digitalization, called Digitaline Nativelle *Intramuscular*, has been introduced by Varick Pharmacal Company, a division of E. Fougera & Company, Inc. It is virtually non-alcoholic, provides an ease of administration, avoids hazards of intravenous injections, and is rapidly absorbed and permits immediate therapy.

A postgraduate course in pediatric allergy, under the direction of Dr. Bret Ratner, Professor of Clinical Pediatrics and Associate Professor of Immunology, at New York Medical College, will be presented at the Flower and Fifth Avenue Hospitals by the New York Medical College. There will be 30 Wednesday sessions, commencing November 4, through May 31, 1954. Applicants must be certified in pediatrics or have the requirements for certification. Group limited. Application should be made to: Dean, New York Medical College, 106th Street and Fifth Avenue, New York 29, New York.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for October 19-31, under the direction of Dr. Paul H. Holinger. Interested registrants will please write to the Dept. of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk St., Chicago 12, Illinois.

The National Gastroenterological Association announces that its Fifth Annual Course in Postgraduate Gastroenterology will be given at the Hotel Biltmore, Los Angeles, October 15, 16, and 17. Dr. Owen H. Wangensteen is the director and co-chairman and Dr. I. Snapper will serve as medical co-ordinator. For further information and enrollment write to the National Gastroenterological Association, Department GSJ, 1819 Broadway, New York 23, New York.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